Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

Kaapstad, 21 Januarie 1961 Deel 35 No 3 Volume 35 Cape Town, 21 January 1961

KEISERSNIT VIR EKLAMPSIE*

'N VOORLOPIGE OORSIG

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In 1922 het Eden[1] die volgende resultate gepubliseer, waar konserwatiewe en radikale behandeling vir eklampsie vergelyk is (Tabel I):

TABEL I. EKLAMPSIE: RESULTATE VAN RADIKALE BEHANDELING (EDEN)

	Ligte gevalle; mortaliteit %	*Ernstige gevalle; mortaliteit* %
Konserwatiewe verlossing ..	5	34
Keisersnit	11	46
Accouchement force ..	18	63

Die skrikwekkende bevinding van 'n moederlike mortaliteit van om-en-by 40% het blykbaar tot vandag toe sy uitwerking laat voel, want Greenhill[2] beweer: 'If there is one thing that is settled in the treatment of eclampsia, it is that Caesarean section routinely performed, gives the highest maternal mortality.'

Menon[3] het in 1,515 gevalle van eklampsie 'n moederlike mortaliteit van 15% gevind. Met die standaard hedendaagse kalmeer-behandeling is die moederlike mortaliteit in die omstreke van 8%.

Alhoewel konserwatiewe behandeling 'n redelike lae moederlike mortaliteit gee, is die perinatale mortaliteit nog baie hoog — tussen 30% en 40% (Dewar en Morris[4]).

Gedurende die laaste 5 jaar is daar 'n effense neiging tot 'n meer liberale gebruik van keisersnit as 'n metode van verlossing. Corkhill[5] van Nieu-Seeland het beweer dat keisersnit die beste behandeling is onder die volgende omstandighede: (1) Waar stuiptrekkings onder beheer is, maar waar kraam nog nie begin het nie; en (2) waar stuiptrekkings nog nie onder volkome beheer is nie, al is die pasiënt in kraam, maar die serviks nog maar min ontsluit is.

TABEL II. MOEDERLIKE PROGNOSE VAN VERSKILLENDE TIPES VAN STUIPTREKKINGS (MENON)

Tipe van stuiptrekkings	*Aantal gevalle*	*Aantal sterftes*	%
Antepartum	826	145	17·5
Intrapartum	61	4	6·5
Postpartum	264	25	9·3

Menon het ook gevind dat antepartum-eklampsie 'n slegter moederlike prognose dra as intrapartum- en postpartum-eklampsie (Tabel II).

Hy het ook gevind dat hoe langer die tydperk is tussen die eerste stuiptrekking en verlossing, hoe hoër die mortaliteit vir beide moeder en baba (Tabel III).

Ten spyte van hierdie belangrike bevindings beweer Kellar:[6] 'There is no real obstetric problem in the management of eclampsia. Until the patient has recovered from her fit or series of fits, the question of delivery does not arise. If labour has not begun some 24 hours after the last fit, the question of inducing labour will arise.'

TABEL III. VERBAND TUSSEN TYDSVERLOOP TUSSEN EERSTE EN TWEEDE STUIPTREKKING EN GEMIDDELDE MORTALITEIT

Tydsverloop tussen eerste stuiptrekking en verlossing	*Gemiddelde mortaliteit* %
0 - 2 uur	7·0
2 - 4 uur	12·8
4 - 8 uur	18·6
8 - 12 uur	22·0
12 - 18 uur	25·0
18 - 24 uur	32·0
Meer as 24 uur	42·0

TABEL IV. PRE-EKLAMPSIE

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
240	Konserwatief ..	239	1	223	17 (7%)
27	Keisersnit	27	0	26	1 (4%)

Dit word algemeen aanvaar dat keisersnit die behandeling vir *dreigende* eklampsie is. In 1957 is 27 keisersnitte in die Karl Bremer-hospitaal gedoen met geen moederlike mortaliteit nie en 'n perinatale mortaliteit van 4% (Tabel IV). In dieselfde tydperk was daar 'n 20% moederlike mortaliteit en 40% perinatale mortaliteit vir eklampsie-gevalle wat dan op konserwatiewe metodes behandel is. Dit is toe besluit om keisersnit te doen vir antepartum-eklampsie om die perinatale mortaliteit te probeer verbeter. Sekere voorwaardes is gestel: (1) Die swangerskap moes 32 weke bereik het, en (2) die uterus moet òf in swak kraam wees, òf glad nie in kraam wees nie.

METODE

Roetine kalmeringsbehandeling is gegee by opname, tensy die pasiënt komateus was. Antibiotika is profilakties gegee.

Hidrokortisoon, 100 mg., is in 1,000 ml. dekstrose en water gegee gedurende en na die operasie; hierdie deel van die behandeling is baie belangrik.

Narkose. Pentotal-induksie; daarna N_2O en O_2 (hoë konsentrasie O_2), met 'n spierverslappingsmiddel — 'flaxe-

* Lesing gelewer by die Kongres van die Suid-Afrikaanse Vereniging van Verloskundiges en Ginekoloë (M.V.S.A.) te Johannesburg in April 1960.

dil' of kurare. Epidurale narkose word nie gebruik nie omdat daar te veel stimulasie van die pasiënt is.

Gedurende die 3 jaar 1957 - 1959 was daar 48 gevalle van eklampsie uit 5,089 bevallings — 'n voorkoms van 0·9%. Sewe gevalle het postpartum-stuiptrekkings gehad. Van die orige 41 gevalle is 21 konserwatief behandel en 20 radikaal (17 keisersnitte en 3 histerotomies). Van die 21 gevalle wat konserwatief behandel is, is die fetale hart in 2 gevalle nie gehoor by opname nie.

EKLAMPSIE: KONSERWATIEWE BEHANDELING

In die 19 gevalle waar die fetale hart gehoor is by opname en konserwatiewe behandeling toegepas is, is 4 moeders dood (20%) en die perinatale mortaliteit was 7 (37%)

TABEL V. EKLAMPSIE (1957 - 1959) KONSERWATIEWE BEHANDELING

Stuiptrekkings		*Fetale hart gehoor*	*Dood-gebore*	*Neonatale dood*	*Moeder-like mortaliteit*	
Antepartum	19	18	5	1	4	(20%)
Intrapartum	2	1	0	1	0	
Postpartum	7	—	—	—	1	
Totaal	28	19	5	2	5	(18%)
			(37%)			

(Tabel V). (Die kortste periode van swangerskap was 37 weke). Vier gevalle het postpartum-stuiptrekkings gehad.

In 17 gevalle is 'n keisersnit gedoen. Daar was geen moederlike mortaliteit nie en net een baba is dood (neonatale dood) — 6% (Tabel VI).

TABEL VI. RESULTATE VAN KONSERWATIEWE BEHANDELING EN KEISERSNIT

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
17	Keisersnit	17	0	16	1 (6%)
3	Histerotomie ..	2	1	1	2
19	Konserwatief ..	15	4 (20%)	12	3 (37%)

In 3 gevalle is 'n abdominale histerotomie gedoen — 2 op 26 weke en 1 op 24 weke van swangerskap. Een moeder is dood (serebrale bloeding), 1 baba is doodgebore, 1 is dood na 6 uur, en 1 baba wat 1 pd. 8¼ oz., by geboorte geweeg het, leef.

Geen gevalle wat met keisersnit behandel is, het post-partum-stuiptrekkings gehad nie en die postoperatiewe verloop van sake was besonder bevredigend!

Eden[1] het sekere maatstawwe neergelê waarvan enige 2 'n geval van eklampsie as ernstig bestempel:

1. Urine-uitskeiding van minder as 800 ml. per 24 uur.
2. Koma wat diep is.
3. Polsspoed van meer as 120 per minuut.
4. Temperatuur van hoër as 103°F.
5. Sistoliese bloeddruk van meer as 200 mm./Hg.
6. Meer as 10 stuiptrekkings.
7. Soliede proteïenurie.
8. Afwesigheid van, of min, edeem.

Nie een van die reeks het 'n temperatuur van meer as 101°F. gehad by opname nie. Die hoeveelheid edeem was ongelukkig in die meeste gevalle nie gespesifiseer nie en is dus buite rekening gelaat. Geen gevalle wat radikaal behandel is, is 24 uur lank voor operasie dopgehou nie, en dus is die urine-uitskeiding ook buite rekening gelaat. Daar was egter heelwat gevalle wat baie min urine in die blaas gehad het by opname.

TABEL VII. SISTOLIESE BLOEDDRUK HOËR AS 200 MM. HG

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
7	Keisersnit	6	1 (14%)	4	3 (43%)
5	Konserwatief ..	4	1 (20%)	3	2 (40%)
12		10	2 (17%)	7	5 (42%)

TABEL VIII. EIWIT SOLIED

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
10	Keisersnit	10	0 (0%)	8	2 (20%)
8	Konserwatief ..	5	3 (38%)	5	3 (38%)
18		15	3 (16%)	13	5 (28%)

TEBEL IX. POLS VINNIGER AS 120

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
10	Keisersnit	10	0 (0%)	8	2 (20%)
8	Konserwatief ..	5	3 (38%)	5	3 (38%)
18		15	3 (16%)	13	5 (28%)

TABEL X. STUIPTREKKINGS MEER AS 10

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
4	Keisersnit	4	0 (0%)	3	1 (25%)
2	Konserwatief ..	1	1 (50%)	1	1 (50%)
6		5	1 (17%)	4	2 (33%)

TABEL XI. KOMA

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
7	Keisersnit	6	1 (14%)	4	3 (43%)
6	Konserwatief ..	3	3 (50%)	2	4 (66%)
13		9	4 (30%)	6	7 (54%)

TABEL XII. NULLIPARITEIT

Gevalle	*Behandeling*	*Moeder*		*Baba*	
		Lewe	*Dood*	*Lewe*	*Dood*
8	Keisersnit	8	0 (0%)	7	1 (12%)
11	Konserwatief ..	10	1 (8%)	7	4 (36%)
19		18	1 (5%)	14	5 (25%)

Koma, polsspoed, bloeddruk, proteïenurie, en stuiptrekkings is vergelyk in gevalle wat *met keisersnit* of *konserwatief* behandel is. Die gegewens in hierdie verband word in Tabelle VII - XI gegee.

Uit die tabelle is dit dus duidelik dat meer van die gevalle wat deur keisersnit verlos is, in die ernstige eklampsie-groep geval het, en ten spyte daarvan is die resultate vir beide moeder en kind aansienlik beter as in die gevalle wat konserwatief behandel is.

Ook is die effek van pariteit in die vorms van behandeling vergelyk en veral in die nullipara, waar kraam met moeite ge-induseer kan word; of, waar kraam van 'n swak tipe is, is die perinatale mortaliteit 3 keer swakker in die konserwatiewe behandelingsgroep (Tabel XII).

Op grond van hierdie voorlopige oorsig wil dit dus voorkom of daar 'n baie groter plek vir keisersnit is — onderhewig aan sekere voorwaardes — in gevalle van eklampsie waar die fetus nog lewe, en dat die swak reputasie van keisersnit onder moderne toestande, nie gestaaf is nie!

SUMMARY

1. The results of treatment in 48 cases of eclampsia are presented.

2. A case is made out for Caesarean section where the foetus is alive and mature enough to survive.

3. Out of 17 Caesarean sections there were no maternal deaths, and 1 child was lost.

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TOLBUTAMIDE OVERDOSAGE AND IRREVERSIBLE CEREBRAL DAMAGE

A CASE REPORT

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Irreversible cerebral damage due to prolonged hypoglycaemia is a well-known result of insulin overdosage. Since the discovery of oral hypoglycaemic agents the possibility has existed that similar effects could be caused by these drugs. Experimental evidence, however, tends to negate this possibility.

In most laboratory animals the hypoglycaemic action of tolbutamide is qualitative rather than quantitative. Large doses do not proportionately increase the degree of hypoglycaemia. There is, however, a difference in the sensitivity of various species. The threshold dose in the dog is one-tenth of that in the rabbit.[1] In dogs prolonged dosage in excess of 100 mg. per kg. causes death from acute hypoglycaemia.[2]

In human diabetics large doses of sulphonylurea derivatives do not significantly increase the response, though there have been reports of severe hypoglycaemia in diabetic patients who have appeared to be unduly sensitive to therapeutic doses of these drugs. McKendry[3] recorded the case of an 84-year-old diabetic man who twice developed hypoglycaemia while on normal doses of tolbutamide. On the second occasion the blood sugar was reduced to 26 mg. per 100 ml. and he succumbed to irreversible brain damage. Coates and Robbins[4] described 'hypoglycaemic shock' in an 88-year-old woman whose blood sugar was reduced to 8 mg. per 100 ml. by therapeutic doses of chlorpropamide. Camerini-Davalos *et al.*[5] recorded 2 patients in whom moderately severe hypoglycaemic reactions resulted from therapy with carbutamide. Seidler *et al.*[6] had 3 patients in whom hypoglycaemia occurred during therapy with carbutamide or tolbutamide. One of these became unconscious. A feature of these reported cases is that all of the hypoglycaemic reactions developed comparatively early in the course of treatment with oral hypoglycaemic agents. It appears also that the tendency to hypoglycaemia may continue for several days following cessation of treatment.

There have been no published reports of the effects of excessively large doses of these drugs in humans. Dr. Henry Dolger of the Mount Sinai Hospital, New York, had a diabetic patient who took 25 g. of tolbutamide in one day and apparently suffered no untoward effects apart from asthenia.[7] The effects of a massive dose of tolbutamide on a healthy subject have never been reported. The following case is probably unique.

CASE REPORT

A 30-year-old Indian woman was brought to the casualty department at Edendale Hospital on a Sunday evening in October 1959. She was deeply unconscious. Relatives stated that she had taken an overdose of tablets which had been supplied to her diabetic mother at this hospital. At this stage the tablets were not identified. The patient's stomach was washed out and she was given 40 ml. of a 50% glucose solution intravenously without any effect on her state of consciousness. She remained deeply comatose and was admitted to hospital at 10.30 p.m. when an intravenous infusion of 5% glucose in water was started. She was a well-nourished woman of average weight. Her blood pressure was 120/80 mm. Hg. There was no cyanosis and respiration was regular and normal. Her pupils were dilated and did not react to light. The tendon reflexes were normal. No other evidence of physical disease was found. The urine was normal.

During the night, because at that stage barbiturate poisoning was considered possible, she was given intravenous injections of bemegride and amiphenazole. Her coma lightened slightly and next morning she responded to painful stimuli and her pupils were smaller and reacted sluggishly to light, but she did not speak. Atropine and penicillin were given because there was excess bronchial secretion. Later methylamphetamine was administered intramuscularly in doses of 15 mg. 8-hourly. On the morning after her admission to hospital her blood sugar was 103 mg. per 100 ml. Her cerebrospinal fluid, serum electrolytes (sodium, potassium and chloride) and blood urea were normal.

She never regained consciousness although she survived for

6 months. Evidence, reconstructed from that gleaned from the patient's relatives and the hospital records, yielded the following story: The patient's mother was a middle-aged diabetic who was being treated with tolbutamide in a dose of 0·5 g. (1 tablet) twice daily. On the Thursday before her daughter's admission she had attended the outpatient department and received a month's supply of 60 tablets. She began using these tablets on that day. She lived with her son and daughter (the patient). Following an unhappy marriage the patient had been deserted by her husband who had taken their children away with him. As a result she had become depressed and had threatened suicide several times.

During that week-end the patient had been alone at home from Saturday afternoon at about 4.30 p.m. until she was discovered unconscious more than 24 hours later. Her mother and brother were closely questioned on several occasions regarding the possible causes of her unconsciousness. Their first and sustained belief was that she had consumed an overdose of the tablets which her mother took for diabetes. This belief was strengthened by the fact that the tablets had been available to her, and that only 12 tablets remained in the container after she was discovered in coma. Efforts were made to establish the possibility of any other form of poisoning. It appeared that the usual kinds of household remedies were present in the house. These included laxatives, aspirins, vitamin pills and potassium permanganate crystals. None of these was missing, nor did the patient's condition suggest poisoning by any of them. There was no insulin in the house. No fire had been burning.

Subsequent Course

During the first 48 hours after admission to hospital the patient's coma lightened slightly, she responded to painful stimuli, her pupils reacted to light and her deep reflexes were brisk and equal. She did not speak, nor did she make any response to questions. Blood-sugar estimations gave normal results. She was incontinent of urine and faeces, and was fed with a gastric tube. It became obvious that she had suffered irreversible cerebral damage. From the first week onwards she lay curled up in bed grinding her teeth and uttering periodic unintelligible shrieks without provocation. Chlorpromazine and later phenobarbitone were used as sedatives. In many respects her condition resembled that of a severe grade of mental defective.

As the months passed she lost much weight despite efforts to maintain her food intake through the gastric tube. She gradually developed a quadriplegia with exaggerated deep reflexes and bilateral ankle clonus. Her pupils retained their reaction to light but it appeared that she had some impairment of vision. She continued to menstruate fairly regularly. Examination of the cerebrospinal fluid 4 months after admission to hospital showed a slight excess of globulin but no other abnormality. She died, after 6 months of progressive intellectual and neurological deterioration, in April 1960.

Postmortem Findings

At the postmortem examination (performed by Dr. P. Matthews) the body weighed 63 lb. There were flexion deformities of the limbs and superficial ulcers over both hips. There was a chronic ulcer, measuring 2 cm. by ½ cm., in the anterior wall of the upper third of the oesophagus. This was presumably caused by the gastric tube. The heart, liver and kidneys were atrophic, being about half their expected size. The bladder showed signs of chronic infection. The brain was oedematous and the lateral ventricles were slightly dilated but there was no other gross lesion. Dr. J. Suskin reported that sections of the brain showed neuronal degeneration and gliosis. These changes are not specific, but would be expected following prolonged anoxia or hypoglycaemia with survival for some time after the accident. Other organs showed no significant changes.

DISCUSSION

Though there is no definite proof that this patient took an overdose of tolbutamide, the circumstantial evidence points strongly to the conclusion that she consumed 20 g. or more of the drug about 24 hours before she was brought to hospital. This represents a dose of approximately 330 mg. per kg. body weight. (Her weight on admission was estimated to be 60 kg.) There can be no doubt that she suffered irreversible cerebral damage with permanent depression of higher cerebral function and diffuse damage to the pyramidal tracts. It is difficult to escape the conclusion that this was due to prolonged hypoglycaemia though, unfortunately, blood was not taken for glucose estimation before intravenous glucose was administered. The clinical and pathological features of the case resembled those of cerebral damage caused by anoxia or hypoglycaemia.

Some animal experiments tend to contradict the conclusion that tolbutamide was responsible, but it appears that different species vary considerably in their response to sulphonylurea drugs. The few recorded cases of severe hypoglycaemia due to therapeutic doses of sulphonylurea drugs indicate that there must also be an individual variation in response to these drugs. Since the action of tolbutamide depends partly on the integrity of the pancreas and its ability to secrete insulin, it is possible that large doses in healthy subjects would have a quantitatively different action to that in diabetics who show a response to the drug. Furthermore it appears that the hypoglycaemic effect is maximal after the first few doses; this is probably due to liberation of insulin from the pancreas in excessively large amounts. It is therefore likely, in the light of present knowledge, that the cerebral damage in this previously healthy patient was caused by an overdose of tolbutamide, probably by means of prolonged hypoglycaemia.

SUMMARY

A case is described in which irreversible brain damage apparently followed a massive overdose of tolbutamide, taken with suicidal intent. Severe hypoglycaemia has occasionally been reported after therapeutic doses of sulphonylurea compounds. There is no previous record of the effect of an overdose in a healthy person.

ADDENDUM

Since this report was submitted for publication, Locket and Brown[8] have described 2 cases of coma which were apparently caused by oral hypoglycaemic agents. In 1 case a non-diabetic woman deliberately took an overdose of tablets and died after being comatose for 96 hours.

I thank Messrs. Hoechst Pharmaceuticals (Pty.) Ltd. for valuable information concerning the toxic effects of oral hypoglycaemic agents. Permission to publish this report was given by Dr. T. M. Adnams, Medical Superintendent, Edendale Hospital, and authority to include the postmortem findings was given by the Chief Magistrate, Pietermaritzburg.

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VAN DIE REDAKSIE : EDITORIAL

ASPEKTE VAN GEESTESGESONDHEIDSDIENSTE

Die algemene jaarverslag van die Suid-Afrikaanse Nasionale Raad vir Geestesgesondheid, wat so pas verskyn het, vestig weer by vernuwing die aandag op die groot en omvattende dienste wat daar in ons land deur hierdie organisasie en sy konstituerende verenigings gelewer word. Dit vestig ook die aandag op allerlei *ander* probleme in verband met die voorsiening van geestesgesondheidsdienste in ons land.

Die Suid-Afrikaanse Nasionale Raad vir Geestesgesondheid het, sedert sy stigting ongeveer 40 jaar gelede, 'n baie belangrike rol gespeel in die daarstelling van dienste vir geestesversteurdes in ons land. Daarnaas was die Raad besonder aktief by die beïnvloeding van die openbare mening om 'n meer aanvaarde houding jeens die probleem van geestesiekte aan te neem. Om die omvang van die dienste wat deur die Raad gelewer word te beklemtoon, sal dit goed wees om sy oogmerke hier weer te gee. Die oogmerke van die Raad is om:

(*a*) Alle sake van 'n nasionale aard in verband met geestesgesondheid asook alle sake van 'n internasionale aard in verband met geestesgesondheid, sover dit op watter wyse ook betrekking het op die belange van die gebied wat deur die Raad bedien word, te behartig;

(*b*) Die geestesgesondheid by die hele gemeenskap, afgesien van ras of geloof, tot die hoogste moontlike peil te bevorder, waarby die uitdrukking ‚geestesgesondheid' in sy ruimste biologiese, geneeskundige, opvoedkundige, beroeps- en maatskaplike sin gebruik word;

(*c*) Die vermoë te ontwikkel om 'n bevredigende en harmoniese lewe in 'n veranderende omgewing te lei. Dit behels beheer van emosionele botsing en spanning by die indiwidu, tussen indiwidue, en tussen groepe;

(*d*) Maatreëls te tref om enige bestaande fasiliteite te help verbeter wat bedoel is om ongesonde geestestoestande te voorkom en te behandel, nasorg te verskaf aan persone wat behandeling ter verbetering van hulle geestesgesondheid ontvang het, en om die persone te rehabiliteer; dit sluit in die behandeling van alle soorte geestesafwykings, of die persoon nou kranksinnig verklaar kan word of nie, en of dit nou by indiwidue dan wel by groepe voorkom; en omvat emosionele en gedragsprobleme, persoonlikheidsgebreke, psigoneurose, dranksugtigheid, epilepsie, afwykinge van verstand, vertraagde opvoeding, wanaanpassings by die gesins-, beroeps- of maatskaplike omgewing, en die psigosomatiese en maatskaplike aspekte van die geneeskunde en verpleging;

(*e*) Die openbare mening met betrekking tot alle geestesgesondheidsake, insluitende die voorafgaande, te help vorm en ontwikkel;

(*f*) Samewerking aan te moedig tussen wetenskaplike en professionele groepe wat iets tot die verbetering van geestesgesondheid bydra of kan bydra;

(*g*) Op die gebied van geestesgesondheid wetenskaplike navorsingswerk te verrig, opnames te maak en demonstrasies te hou, en om sulke bedrywighede te bevorder;

(*h*) 'n Hoër peil van opleiding vir alle groepe op die gebied van geestesgesondheid te bepleit;

(*i*) Doeltreffende samewerking met en tussen staatsdepartemente, provinsiale administrasies, munisipaliteite, welsynsorganisasies, inrigtings, verenigings en indiwidue in verband met geestesgesondheidsake of enige aspek daarvan te handhaaf en te bevorder;

(*j*) Die stigting en onderhoud van geestesgesondheidsverenigings aan te moedig en te bevorder en om hulle bedrywighede te koördineer en leiding in verband daarmee te verskaf; en om

(*k*) As amptelike skakelliggaam tussen die samestellende liggame aan die een kant en Staatsdepartemente en nasionale liggame aan die ander kant op te tree.

As konstituerende dele van die Nasionale Raad vir Geestesgesondheid is daar die verskillende verenigings vir geestesgesondheid wat versprei is in al die grotere sentrums in ons land. Daar is naamlik sulke verenigings in Johannesburg, Pretoria, Potchefstroom, Durban, Pietermaritzburg, Kaapstad, Oos-Londen, Port-Elizabeth, Kimberley, en Bloemfontein. Hierdie verenigings verrig elkeen in sy eie gebied uitgebreide geestesgesondheidsdienste, insluitende die hou van klinieke, die organisasie van gevallestudiewerk, nasorgsdienste, ens.

In 'n land soos Suid-Afrika waar daar op 'n praktiese vlak nog so 'n groot behoefte bestaan aan die lewering van genoegsame en bevredigende geestesgesondheidsdienste, kan ons nie die lofwaardige pogings van hierdie organisasie genoeg prys nie. Langs hierdie weg wil ons dan ook ons heelhartige ondersteuning aan die organisasie toesê.

In die algemeen sou ons kon sê dat die probleme wat betref die lewer van geestesgesondheidsdienste legio is. Ons kan en wil nie nou na almal verwys nie. Wat ons egter wel wil sê is dat die grootste behoefte waarskynlik nog op die gebied van mannekrag is. Daar is nie genoeg geneeshere wat in die psigiatrie belangstel en gekwalifiseer is, om die werk wat dringend gedoen moet word, te doen nie. Uitbreiding van fasiliteite val dus weg as die basiese verpligtinge nie eers nagekom kan word nie.

Om die leemte aan te vul, sal dit nodig wees om meer fasiliteite aan al ons mediese skole te skep vir die opleiding van psigiaters. In die verlede het studente nie in die vak belanggestel nie — veral nie met betrekking tot verdere nagraadse studie nie — hoofsaaklik omdat die psigiatrie te ver verwyderd gestaan het van die algemene medisyne.

Dit sal dus nodig wees om die vak meer as in die verlede te integreer met die algemene medisyne. Daarna sal dit nodig wees om meer studente op 'n gevorderde vlak op te lei sodat hulle weer as onderwysers van die vak kan optree. Eers dan kan ons dink aan radikale uitbreiding van fasiliteite soos die oprig van hospitale vir senuweekwale, ens. Dit is dus by die opleiding van studente aan die mediese skole dat ons moet begin. Intussen lewer 'n organisasie soos die Suid-Afrikaanse Nasionale Raad vir Geestesgesondheid, tesame met sy konstituerende verenigings, 'n onberekenbare diens op die praktiese vlak, ook en veral as 'n skakeldiens met die publiek.

SNAKES, SPIDERS AND SCORPIONS

The incidence of snakebite in Southern Africa increases gradually with the return of warm conditions and increased rainfall, being at its lowest in the months of June, July, and August.[1] A true estimate of the incidence is difficult to obtain despite the requirement of the law that all cases of snakebite, whether fatal or not, be reported to a magistrate for the purpose of medical statistics.

The majority of those bitten have been of the male sex. Among White persons it is interesting that children up to ten years of age represent about 25 per cent of those who are bitten, presumably because they usually are barefooted at that age. The incidence of snakebite could probably be halved if shoes or boots were worn.[2]

The puff-adder is the largest and most venomous viper in Southern Africa, and is the commonest cause of snakebite, followed by the night-adder. A small number of cases is due to the ringhals, the Cape cobra, and the mamba. The incidence of snakebite is highest in the coastal regions especially in Natal, but also in the Transvaal lowveld and near large centres in the highveld.

Persons bitten by the ubiquitous adders seldom suffer much harm, but sometimes there are serious consequences. Death is delayed but uncommon; recovery is usually complete after viper bite, even if it is sometimes slow. The venom may cause marked damage at the site of the bite, but its effect on more distant structures appears late if at all. Following a bite from an elapine snake (cobra, ringhals, mamba) general symptoms may come on after a period varying from minutes to hours. This depends on the species, the dose of venom, and presumably on factors inherent in the patient.

If specific antivenom is available it is unquestionably the best treatment for snake-bite. To ensure rapidity of action it should be given intravenously after utilizing any method available to extract some of the venom from the wound, and, in the case of elapine snakes, after the earliest possible application of a ligature to prevent the entry of the venom into the blood stream. The bite is almost invariably on an extremity; a tourniquet should be applied well above the site, and it should be loosened for five to ten seconds every ten to fifteen minutes. The usefulness of this procedure has been questioned in the case of bites by vipers. As antivenom should be administered as soon as possible, the injection should usually be made without first giving a trial dose. In the circumstances it might be advisable to give 1 ml. of 1 : 1,000 adrenaline intramuscularly as soon as the serum injection is begun, and to repeat the dose of adrenaline, or half of it, fifteen minutes later. The dose of serum should be large and never less than that advised in the leaflet of the manufacturer, whose instructions should be followed. The time elapsing between the bite and the injection of serum is also an influencing factor on dosage.

If intravenous injection is impracticable because the patient is in a state of collapse, the intraperitoneal route has been suggested, or failing that, the intramuscular. Local injections of 2 - 3 ml. have also been advised if the patient is treated soon after he is bitten. This is specially indicated in viper bites, when even larger doses are recommended if anatomically possible.

The amount of any venom which will kill young children is, owing to their very small body-weight, much less than that causing fatality in adults. The dose of antivenom injected into children should be related to the presumed dose of venom injected rather than to their body-weight, and should, therefore, *never be less* than the adult dose.

Tetanus antitoxin should be given to all cases of snakebite because of the possibility of tetanus infection as a complication. There may be an appreciable risk even when the snake is non-poisonous.[2]

Certain snakes are 'spitters', that is to say they can eject venom which may strike the eyes causing intense irritation and inflammation. In such cases the eyes should be washed with water or other harmless fluid, and diluted antivenene may then be instilled; antivenene should not be injected into the victim.

The female of certain Latrodectus species of spider, commonly called 'knopie-spinnekop' or 'button-spider' can produce serious illness in man.[2] This spider is about 20 mm. long and has a well-rounded belly from which it derives its name. It is jet-black or dark-brown in colour, and the dorsal surface may have irregular white or yellow spots. Bites have been reported from the wheatlands of the Western Province and elsewhere. The potent venom can produce excruciating cramp-like pain in the limbs, chest and abdomen of the victim. On occasion the syndrome may resemble an acute-abdominal condition. The introduction of a specific antiserum has revolutionized the treatment, and remarkable recoveries follow injection of this material. During 1949 to 1953, 1,794 ampoules were issued by the South African Institute for Medical Research, Johannesburg.

The toxicity of scorpion venom would appear to be much greater than that of snake venoms, but the scorpion injects only a small amount of venom.[4] If treatment is deemed necessary the measures include the use of a tourniquet, loosened for one minute every ten minutes, artificial respiration with oxygen (if respiration has been depressed), injection of specific antiserum (if this can be obtained), control of convulsions (the use of central nervous depressants requires great care), and the administration of calcium gluconate injection to relieve muscle cramps. Application of crushed ice with a little water to the site of the bite will delay the onset of symptoms; an entire limb can be immersed in this cold mixture for about two hours. Severe allergic episodes are best treated by giving hydrocortisone injection by slow intravenous infusion.

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URINARY CALCULI AND SERUM CALCIUM LEVELS IN AFRICANS AND INDIANS

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Among the White, Indian and Coloured people in South Africa the incidence of urinary calculus formation is similar to that in Europe. In the African population the disease is rare; this has been confirmed by several observers.[9-12]

In an analysis of over 1,000,000 records in African mine-workers, Orenstein[12] found only 1 case in which a clinical diagnosis of a renal calculus had been made. Vermooten[16] analysed the admissions to the Johannesburg Non-European Hospital from 1922 - 1935 and found only 4 cases in 91,000 admissions. Only 1 of these was in an African, a woman in whom a ureteral calculus was suspected but never proved.

Lopis and Kaplan[9] recorded the first proved case in 1948 in an African, a Shangaan born in Beira. Muskat[11] recorded 2 further cases in 1951. One was a Rhodesian-born African and the origin of the other patient was not stated.

In 1957 Politzer and Beuchat[13] reported the case of a Shangaan male patient from Portuguese East Africa with vesical calculi. Gelfand,[5] discussing Rhodesian Africans, stated that calculi do occur but gave no clinical details.

It is noteworthy that to date most recorded calculi in Africans have occurred in those originating north of the Union's borders, where calculi appear to be more common.

In King Edward VIII Hospital, Durban, the records of all Africans admitted from 1 January 1951 to 30 June 1959, a period of 8½ years, have been analysed. Of 483,450 admissions there were 7 cases of urinary calculi in locally-born Africans, all men. This represents an incidence of 0·0014%. By way of contrast, out of a total of 9,600 Indian admissions in 1 year, there were 12 cases of proved calculi representing an incidence of 0·125%.

Serum-calcium levels were estimated in control groups of White, African and Indian patients. The method of estimation was that of Bett and Fraser,[1] using the fluorescent dye 'calcein' and titrating with E.D.T.A. The estimations were undertaken by one technician well versed in the technique, and each estimation was duplicated using standard solutions checked before use. Mixed batches of sera from all groups of patients were estimated simultaneously for calcium levels on the particular day, and therefore it is unlikely that any differences are due to differences in technique from day to day.

The serum-calcium levels of the following groups of patients were estimated:

(1) Indian patients with renal or ureteric calculus, (2) three African patients with vesical calculus, (3) Africans recumbent for some weeks for incidental diseases other than renal, and (4) African paraplegics (Table 1).

In addition, serum-mucoprotein levels were estimated using the turbidimetric method of De la Huerga *et al.*[6] in which the entire mucoprotein complex is measured.

The following are the case records of the 7 African males in whom urinary calculi were diagnosed between 1 January 1951 and 30 June 1959.

CASE RECORDS

Case 1

J.Y., African male (Zulu), aged 69 years. Admitted 3 July 1951, complaining of difficulty with micturition for many years and a recent urethral bleeding episode — about a cupful of blood not mixed with urine. Cystoscopy revealed sand-patches of bilharziasis and several small vesical calculi. No analysis of these was done.

Case 2

A.N., African male (Zulu), aged 25 years. Admitted 28 March 1953, having fallen from the roof of a house and sustained compression fractures of D12 and L1 vertebrae with paraplegia. A laminectomy and suprapubic cystostomy were performed with insertion of a de Pezzer catheter.

While in a wheel-chair, 3½ years later, he was struck by a motor car, sustaining fractures of his pelvis and left femur. The X-ray of the pelvis revealed a large vesical calculus which was subsequently removed by cystostomy on 4 February 1957.

Case 3

M.C., African male (Zulu), aged 37 years. Admitted 4 March 1955, having been run over by a lorry. He was extremely shocked and had bilateral fractures of both horizontal and descending pubic rami and a rupture of the membranous urethra. After resuscitation, a laparotomy was performed and an indwelling urethral catheter and a suprapubic de Pezzer catheter were inserted. On 22 July 1955 a large periurethral abscess was drained.

Four months later he was re-admitted for urethral dilatation and on this occasion a vesical calculus was felt with the end of the urethral dilator. A vesical calculus was removed suprapubically shortly afterwards.

Although advised to return for urethral dilatations, he neglected to do so and his next admission was on 20 May 1958. This was to a medical ward for investigation of a pyrexia of unknown origin. Urine examination revealed pus cells +++++ and a growth of *B.coli* sensitive to polymyxin.

The blood urea was 36 mg. per 100 ml. An intravenous pyelogram (IVP) showed a bilateral hydronephrosis. The urinary infection was treated and, after subsequent admissions, he was transferred to the surgical wards for urethral dilatation. A repeat IVP on 7 October 1958 for upper abdominal pain revealed bilateral staghorn calculi. The blood urea was 45 mg. per 100 ml. On 11 November 1958 the right kidney was explored. The perinephric tissues were adherent and the surface of the kidney was studded with small abscesses. The stone was removed in pieces by pyelolithotomy and nephrolithotomy and the pelvis drained. It is planned to remove the other calculus shortly.

Case 4

M.D., African male (Zulu), aged 76 years. Admitted 5 May 1956 with acute retention of urine for 1 day. Straight X-ray of the pelvis revealed calcification of the bladder wall but, although urine examination revealed pus cells ++, no ova of *S. haematobium* were recovered. The prostate was normal on palpation.

He was re-admitted on 7 September 1957 complaining of dysuria. Straight X-ray of the pelvis revealed calcification of the bladder wall and several dense opacities. Cystoscopy was performed on 12 September 1957 when small vesical calculi were visualized. Some were crushed with a lithotrite and the remainder were removed on 18 September 1957 by suprapubic cystostomy.

Case 5

M.N., African male (Zulu), aged 70 years. Admitted on 28 January 1958 complaining of frequent difficult micturition for 2 months and inability to pass urine for 1 day. Investigations revealed a moderately enlarged prostate. Urine examination showed no pus cells and was sterile. An IVP revealed a

rounded calculus within the left kidney. Transvesical prostatectomy was performed for a fibro-adenomatous hyperplasia of the prostate. The calculus was not removed.

Case 6

Z.D., African male (Zulu), aged 45 years. Admitted on 24 March 1958 complaining of lower abdominal pain, frequency, difficulty with micturition, and perineal pain, for 5 months. When the prostate was palpated he voided urine containing threads of pus. The urine examination revealed pus cells +++ and a moderately heavy growth of *B. proteus* and *Ps. pyocyaneus*. Straight X-ray of the pelvis revealed a large opacity in the bladder. Subsequently a large vesical calculus was removed by suprapubic cystostomy.

Case 7

S.T., African male (Zulu), aged 59 years. This patient was first admitted on 12 October 1956 for investigation of frequency of micturition. On rectal examination the prostate felt normal to palpation. A stricture was found in the bulbous urethra and this was dilated. Cystoscopy revealed only some trabeculation of the bladder.

He was re-admitted on 31 July 1958. He then stated that following the urethral dilatation he was well for several months, but his initial complaints recurred and had increased in severity up to the time of his admission. In addition he had been unable to walk for a week. Examination showed that he had a paraplegia and chronic retention of urine with overflow. Investigation culminated in the removal of an intradural neurilemmoma at the level of D5 - D7. He was discharged, being able to walk again.

His next admission was on 5 May 1959 when he complained of dysuria. An IVP revealed bilateral hydronephrosis and a large filling defect in the bladder consistent with an enlarged prostate.

The blood urea was 24 mg. per 100 ml. Cystoscopy showed a bladder calculus, enlargement of the middle and both lateral lobes of the prostate and marked trabeculation. This was followed by transvesical prostatectomy and removal of the bladder calculus.

The stone was circular in outline and flattened from side to side. It weighed 300 mg. and was a dirty-white colour and firm in consistency.

DISCUSSION

Of the 7 African cases presented here, 1 appears to be a primary urinary calculus (case 6), since no predisposing factors could be found. In 4 of the remaining patients (cases 2, 3, 5 and 7), there was either prolonged recumbency or urinary obstruction, or a combination of both; while in the other two patients (cases 1 and 4), there was associated bilharzial infection of the bladder.

The problems of primary urinary calculi and the complication of recurrent calculus formation therefore seldom occur in the African.

Bilharziasis

The rôle played by bilharzial infection appears, in Africans, at any rate, to be of no major importance. Bilharziasis is endemic in the coastal belts of Natal. Dormer[4] found that 10% of all African school children investigated in Durban have active bilharziasis, which suggests that at any one time the greater part of the local African population is suffering from, or has suffered from, the ravages of this disease. Were it of major significance in calculogenesis more urinary calculi would undoubtedly be found.

On the other hand, urinary bilharziasis is as frequently seen, if not more so, in Indians. The incidence of primary urinary stone in these patients is not very different from that in White patients, while secondary stone formation, particularly ureteric calculus, associated with stricture of the ureter, is one of the commonest urinary diseases encountered.

Predisposing Causes

When calculi do occur in Africans the important predisposing factors are urinary obstruction and infection, and prolonged recumbency, usually due to paraplegia. It should be stressed, however, that all these conditions are common in the African surgical wards, yet urinary calculi are rarely found.

It is unlikely that the rarity of calculus in the African is due to any racial immunity; the American Negro, who springs from the same stock, displays no such immunity,[3] although there is a lower incidence in Negroes than in White Americans. The US Negro has been shown to have a higher level of protective urinary colloids than the White, and Butt has reported an inverse proportion between the incidence of stone and the level of urinary colloids.[2]

Vermooten[17] examined 1,060 pairs of kidneys at the medico-legal laboratories in Johannesburg and found Randall's plaques in 4·3% of Africans, in none of whom calculi were present. The Caucasian incidence of Randall's plaques was 17·2%. This high incidence of Randall's plaques in Africans associated with so small an incidence of calculus, suggests that factors in addition to these plaques are necessary for calculus formation.

Of the environmental factors associated with calculus formation, diet is probably the most significant. Not only are the types of diet and the dietary deficiencies important, but equally so are the secondary metabolic changes induced by such deficiencies. These may be discussed under the following headings:

1. *Calcium Intake*

The majority of urinary calculi are calcium-containing stones. The African's intake is ½ - ⅓rd of the European's and this apparently produces no harmful effects.[17]

2. *Calcium Absorption*

Not only is the intake of calcium lower but in addition absorption may be lessened by 2 factors: (*a*) Vitamin-D deficiency, and (*b*) intestinal hurry. Studies on faecal excretion show that the faecal bulk in Africans is 4 times that of the European and its rate of passage through the alimentary tract is twice as fast.[17]

3. *Urinary Output*

Metabolic studies have shown that the average urinary output of the African is considerably greater than that of the European. This is not a racial characteristic but occurs also in Europeans on a high carbohydrate diet.[18]

4. *Acid-ash Diet*

Vermooten[16] believes that the acid-ash residue produced by a high carbohydrate diet favours the low incidence of urinary calculi in the African.

5. *Vitamin-A Deficiency*

This has been quoted as a factor in the aetiology of urinary calculi.[10] Whether or not this is of any importance, biochemical studies have shown that the blood-levels of vitamin A in the African are within the limits[9] of normal for White patients.

6. *Citrate Theory*

Citrate combines with calcium to form a soluble complex and a reduction in citrate excretion has been suggested as a factor in the production of calculi. Muskat[11] pro-

pounded an interesting theory which suggested that the low incidence of calculi may be related to nutritional liver damage, which is common in the African. This view is based on the observation that oestrogens enhance citrate excretion in the urine and that a damaged liver fails to metabolize circulating oestrogen completely.

7. Serum Proteins and Serum Calcium

The serum-calcium level is independent of the dietary intake; it is, however, partly dependent on the level of the serum proteins. Each gram of albumin and globulin binds approximately 0·84 mg. of calcium as a proteinate.[7] The level of serum proteins is generally found to be lower in the African than in the European[14] and Walker *et al.*[19] showed that the serum-calcium level is, on the average, 1 mg. per 100 ml. lower in the African than in the European and the urinary calcium is considerably less.

Table I shows the mean values of serum calcium obtained for Whites and Indians, which are very similar,

TABLE I. SERUM CALCIUM (MG. PER 100 ML.)

Group	No. of cases	Mean	S.D.	p*
Indian control	34	10·5	0·86	<·05
African control	30	9·9	1·24	
Indian control	34	10·5	0·86	<·05
Indian calculus	30	11·1	1·24	
African control	30	9·9	1·24	<·01
African paraplegics	10	11·1	0·84	
African control	30	9·9	1·24	<·07
African recumbents	18	10·01	0·63	

* t-test

and that for Africans, which is 0·6 mg. less than the Indian level. This difference is probably significant and tends to confirm Walker's findings.[19] The difference between the Indian control and Indian calculus group (0·6 mg.), is probably statistically significant; the difference

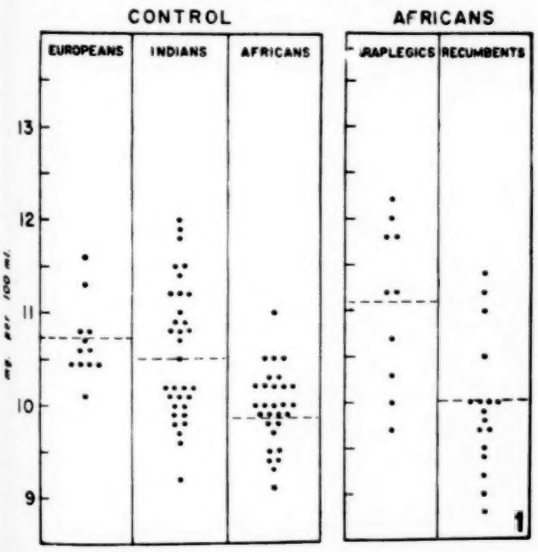

Fig. 1. Distribution of serum calcium levels in sick Africans and control groups. Horizontal dotted lines indicate mean levels in these groups.

between the African control and African paraplegics (1·2 mg.), is likewise significant, while the difference between the African controls and African recumbent patients (0·11 mg.), is not significant (Table I). Fig. 1 shows the distribution of serum-calcium levels in these groups.

In the Indian calculus group, 7 patients had serum-calcium levels above 11·5 mg. per 100 ml. Two of these patients had a serum-calcium level, a year after operative removal of urinary calculi, of 10·8 mg. and 10·2 mg. per 100 ml. respectively. A possible cause for the drop in calcium level in these 2 patients was investigated by comparing samples drawn from a control series at different periods of the same day (Table II). These patients were in hospital for investigation of diseases other than calculus or urinary conditions. There appears to be a significant decrease in calcium levels taken in the middle of the day,

TABLE II. SERUM-CALCIUM LEVELS AT DIFFERENT PERIODS OF THE DAY IN 19 CONTROL PATIENTS WITHOUT CALCULUS OR URINARY DISEASE

	Serum calcium (mg. per 100 ml.)		
Case no.	*Before breakfast*	*1 hour after lunch (1 p.m.)*	*3 - 4 hours after lunch (5 p.m.)*
1..	11·5	10·2	11·4
2..	10·8	10·6	10·6
3..	10·6	10·0	11·1
4..	11·2	10·0	10·5
5..	10·4	10·6	10·7
6..	11·2	11·0	11·4
7..	10·7	10·3	10·4
8..	10·4	10·7	10·1
9..	10·7	10·4	10·5
10..	10·0	9·5	10·7
11..	10·4	10·5	11·3
12..	10·8	10·6	10·7
13..	10·4	10·4	10·4
14..	11·2	10·7	11·0
15..	10·7	10·8	10·9
16..	10·4	10·9	11·0
17..	10·7	10·4	11·0
18..	10·5	10·4	10·3
19..	11·0	10·5	10·5
Mean	10·72	10·45*	10·76
SD	0·20	0·25	0·45
SE	0·05	0·06	0·11

SD = Standard deviation, and SE = Standard error.

* Friedman 2-way analysis of variance[15] shows P < ·02.

after lunch, compared with the fasting morning and late afternoon levels, the mean drop being 0·3 mg. per 100 ml. (with a range up to 1·3 mg. per 100 ml. difference). This may in part explain the drop in these 2 patients on whom repeat estimations were done as out-patients in the middle of the day, their first samples having been taken in hospital in the early morning. The possibility of hyperparathyroidism is still being investigated in the remaining 5 patients.

Serum-mucoprotein levels were estimated in both Indian and African control groups by the turbidimetric method of De la Huerga *et al.*[8] in which the entire mucoprotein complex is measured. The values obtained are shown in Table III. The difference between the Indian and African control figures are not statistically significant (Mann-Whitney U-test and t-test[16]). However the difference between the Indian control and the normal US male levels

TABLE III. SERUM MUCOPROTEIN (MG. PER 100 ML.)

Group	*No. of cases*	*Mean*	*SD*
Indian control	19	51·7	14·8
Indian calculus	22	60·2	36·2
African control	16	68·4	31·7
African paraplegics ..	8	89·8	31·6
Normal US male ..	40	81·2±18·2*	
Normal US female ..	30	77 ±14	

* Normal American figures (De la Heurga *et al.*[5]).

and between the African control and the normal US male levels are both significant at the 0·1% level (t-test).

Further studies are required to elucidate the rôle, if any, of the serum-mucoprotein level in the genesis of renal calculus.

CONCLUSIONS

The explanation for the rarity of primary calculus formation in Africans is not readily available. Calcium intake is lower and faecal calcium output is higher in the African, who usually partakes of a high carbohydrate diet. This, together with a high rate of citrate excretion associated with the liver dysfunction found in chronic malnutrition, may be part of the explanation. It is noteworthy however that, considering the high incidence of urinary infection, obstruction and urinary bilharziasis, secondary stone formation seldom occurs in Africans while it is frequent in Indians.

SUMMARY

1. Seven cases of urinary calculus in Africans are presented.

2. The incidence of urinary calculus in Africans at the King Edward VIII Hospital supports the view that urinary stones seldom occur in this racial group. The problems of primary renal calculus and recurrent calculus formation rarely arise in the African.

3. When calculus does occur in Africans there is nearly always some obvious predisposing factor such as urinary obstruction, bacterial infection and recumbency.

4. Predisposing factors to secondary urinary calculus formation, viz. bilharziasis, long-standing chronic infection, and urinary obstruction are equally common in Indian and African patients. Yet the incidence of both primary and secondary calculus formation is high in the Indian (0·125%) compared to the African (0·0014%).

5. Some of the metabolic factors responsible for calculus formation are discussed.

6. Serum-calcium levels in an African control group show a mean value of 0·6 mg. per 100 ml. less than in Indian controls, a finding similar to that reported between Africans and Whites.

7. A significant variation has been noted in serum-calcium levels taken at varying intervals in relation to meals; in comparing serum-calcium levels, estimations should be done on samples taken at the same time each day bearing in mind that midday samples show a significantly lower level.

8. There appears to be a significant difference between serum-mucoprotein levels in African and Indian control groups and those in US normal males.

Our thanks are due to Mr. S. E. Cruise, Senior Lecturer, Department of Mathematics and Statistics, University of Natal, for the statistical work; and to Mr. C. J. Lockett and Mr. M. Moodley for technical assistance.

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HEREDITARY SPHEROCYTOSIS COMPLICATED BY THE INSPISSATED BILE SYNDROME

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Obstructive jaundice is uncommon in early infancy. It is characterized, as in later life, by enlargement of the liver and spleen, pale acholic stools, heavily bile-stained urine and accumulation of bilirubin glucuronide in the serum. Congenital obliteration of the bile ducts causes about two-thirds of the cases of obstructive jaundice in infancy. Much less common is obstruction from pressure on the biliary tract by cyst, tumour or glands. In one-third of cases, however, the biliary tract is normally formed and there is no evidence of extrahepatic biliary compression. Here, obstruction to flow of bile occurs in the liver through an ill-understood mechanism — accumulation of thickened secretions, swelling or necrosis of liver cells, excessive intrasinusoidal haematopoiesis, an immature biliary system, and dehydration, being variously incriminated.[1] Describing this picture in 1935, Ladd[2] used the term 'inspissated bile syndrome' for the first time.

In contrast to congenital atresia of the bile ducts, where jaundice gradually but steadily deepens and the outcome is invariably fatal unless surgical correction can be achieved, the inspissated bile syndrome is characterized by early onset of severe jaundice and a generally favourable outcome, provided that surgery is not attempted.[3]

The inspissated bile syndrome has often been found to complicate haemolytic disease of the newborn. Hsia *et al.*[4] in their well-known analysis of 156 cases of prolonged obstructive jaundice in infancy, listed 23 cases of erythroblastosis foetalis with this condition. Stempfel *et al.*[1] found

7 infants with obstructive jaundice among 83 cases of erythroblastosis due to rhesus incompatibility, suggesting that the complication may be commoner than was first thought. Presumably it may also result from ABO haemolytic disease.

The syndrome may also occur without evidence of haemolysis or other known cause of jaundice. In such cases, characteristic appearances, suggesting viral infection, are found on microscopic examination of the liver.[3,5] The term 'neonatal hepatitis' has been used for this picture. Recently Hsia *et al.*[6] analysed 59 families in which one or more offspring were affected with neonatal hepatitis. They point out that there may well be a hereditary component in the pathogenesis of this disease.

I found no references to other causes of the inspissated bile syndrome in the literature. The occurrence of this syndrome in a case of hereditary spherocytosis may therefore be of interest.

CASE REPORT*

History

Colin K. was born on 12 February 1957, the first child of healthy unrelated parents. One of his father's brothers and 2 sisters were known to have acholuric jaundice and his paternal grandfather had symptoms suggestive of this condition (Fig. 1). The father was not available for testing at the time of this child's illness but was later found to have spherocytosis and slightly increased osmotic fragility of the red cells, though free of symptoms.

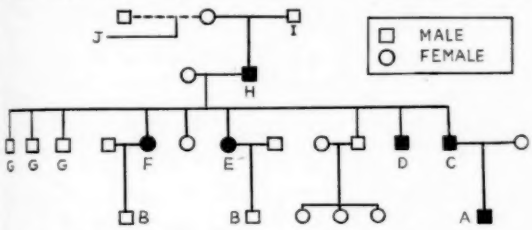

Fig. 1. Family tree of Colin K.

A = Colin K.
B = Clinically normal at birth.
C = Asymptomatic. Spherocytosis and increased fragility.
D = Spherocytosis. Splenectomy.
E = Spherocytosis. Anaemia.
F = Spherocytosis. Splenectomy. Gall stones.
G = Died of unrelated causes. (?)
H = Anaemia. Ulcers on legs.
I = Died in accident. (?)
J = Offspring of second marriage all normal.

The infant was delivered by forceps for disproportion at full term after a normal pregnancy. His weight at birth was 7 lb. 11 oz. He appeared normal at first but slight jaundice was noted at 4 hours, gradually becoming more intense. The liver and spleen were much enlarged. Stools consisted initially of dark meconium but changed to brown on the third day; thereafter they were clay-coloured. The urine became progressively more bile-stained. The jaundice deepened and the haemoglobin dropped rapidly to 8·5 g. per 100 ml. A simple transfusion of whole blood was given on the 12th day. There was considerable feeding difficulty and frequent vomiting, various formulae being tried.

Examination

The infant was first seen by me when he was 4 weeks old. Examination revealed an undernourished, chronically ill, icteric infant, the skin and mucosae being greenish-yellow in colour. He was only 1 oz. over his birth weight. No cataracts or retinitis were noted on ophthalmological examination. The liver was much enlarged and firm and smooth; the lower margin being felt 3 fingerbreadths below the costal margin. The spleen was easily palpable. No gastric peristalsis was visible and no other masses were detected.

Blood Investigations

RBCs, 3·2 million per c. mm.; haemoglobin 11 g. per 100 ml. (14·8 g. per 100 ml. = 100%); WBCs 8,200 per c. mm. with neutrophils 50%, lymphocytes 40%, monocytes 7%, and eosinophils 3%. There was some macrocytosis and there were small numbers of normoblasts. No microspherocytes were seen. The reticulocyte count was 3·5%. Saline red-cell fragility was within normal limits (Creed's method[7]). The blood groups of the mother and infant were ORh positive (D). The direct Coomb's test was negative. The serum bilirubin was 17·6 mg. per 100 ml. The Van den Bergh test gave an immediate positive direct reaction. The Kahn test was negative, and blood culture was sterile.

The stools were clay-coloured and the urine deeply bile-stained. Urobilin was found on some occasions. No galactose was present in the urine and repeated examination failed to reveal cells containing inclusion bodies. Studies on red-cell fragility in saline solutions were repeated, yielding a pattern within normal limits (Creed's method[7]).

Treatment

Instillation of concentrated magnesium sulphate solution into the duodenum produced several stools which were pale green in colour. This was repeated for 10 days without diminution in the bilirubinuria or the icterus. Some initial improvement in the feeding was not maintained later and the procedure was abandoned.

On these findings it was considered that atresia of the bile ducts was unlikely, the diagnosis resting between hereditary spherocytosis with a superimposed inspissated bile syndrome, and neonatal hepatitis. The persistent absence of spherocytes and the saline-fragility tests appeared, however, to militate against the former diagnosis. At 6 weeks of age treatment with cortisone was commenced, first by intramuscular injection (12·5 mg. *t.d.s.*) and later by mouth. There was immediate improvement in feeding and weight gain, but no diminution in the jaundice for the first month of treatment. By the sixth week of treatment, however, (age 3 months) the icterus had almost disappeared and the urine was clear during the day, though bile-stained on rising. The liver and spleen were still enlarged.

Progress

The child was not seen again until the age of 7 months. The mother then stated that his progress had been most satisfactory and that he appeared normal in all respects. His weight was 15 lb. 12 oz. and he showed good development. There was well-marked pallor but no jaundice. Two lower incisors had erupted and these were green in colour, a feature noted later in all the deciduous teeth. The liver was soft and of normal size, but the spleen could be felt 1 finger-breadth below the costal margin.

On this occasion the following results were obtained on laboratory investigation: Haemoglobin 8·6 g. per 100 ml., RBCs 3·1 million per c. mm., WBCs 12,600 (differential count normal), no spherocytes or other abnormal cells seen, total protein 5·2 g. per 100 ml., albumin 3·0 g. per 100 ml., α_1 globulin 0·34 g. per 100 ml., α_2 globulin 0·60 g. per 100 ml., β globulin 0·53 g. per 100 ml., fibrinogen 0·27 g. per 100 ml., gamma globulin 0·47 g. per 100 ml., bilirubin glucuronide absent, total serum bilirubin 0·8 mg. per 100 ml., thymol turbidity 2 units, thymol flocculation 0, serum colloidal gold 0, zinc sulphate turbidity 2 units.

No improvement in the anaemia followed a course of intramuscular iron. At 10 months of age the haemoglobin had fallen to 5·4 g. per 100 ml. and the RBCs to 2·1 million per c.mm. The reticulocyte count was 6·8%. The erythrocytes were hypochromic and microcytic with considerable variation in size and shape. Polychromasia was prominent and there were 4 normoblasts per 100 leucocytes. No spherocytic cells were seen. A blood transfusion of 200 ml. of packed cells was given, raising the haemoglobin to 14·0 g. per 100 ml. At 11 months the haemoglobin had again dropped to 9·4 g. per 100

* Reported in brief in a paper presented at the Congress of the Medical Association of Southern Rhodesia, Bulawayo, September 1958.

ml. On this occasion a few microspherocytes were seen in a film of the peripheral blood. There was a reticulocytosis of 7%.

At 1 year of age the osmotic fragility test was repeated by Creed's method, and this time a slightly increased fragility to hypotonic saline solutions was demonstrated. The haemoglobin had dropped to 7·5 g. per 100 ml. and a further transfusion was given.

Splenectomy

The diagnosis of hereditary spherocytosis was at last considered established, and in view of the severity of the anaemia, splenectomy was performed at 13 months after adequate preparation. This procedure was withstood without incident and subsequent progress had been quite satisfactory, the haemoglobin being maintained at normal levels.

Microscopic examination of the spleen revealed no evidence of congestive splenomegaly, 'the picture being consistent with that of acholuric familial jaundice'.

At the time of writing the patient is 2 years 11 months of age and normal in every way apart from green discoloration of the teeth. Infections have not been a problem to date. A recent fragility test using the incubation technique revealed a normal pattern and no spherocytes were noted in the peripheral blood.

DISCUSSION

The occurrence of hereditary spherocytosis in infancy has recently been the subject of 2 excellent reviews.[8,9] The condition has been recorded before the age of 6 months in 40 patients,[9] but in less than 20 did symptoms become manifest in the first 2 weeks of life. In these neonatal cases, jaundice was a frequent feature, contrasting with older subjects where it is not often found. In 3 infants the jaundice was sufficiently severe to produce kernicterus[10-12] and exchange transfusions were performed to lower the serum bilirubin.[10,11,13] There is, however, no report of jaundice of the obstructive type such as in the case here described.

Although no liver biopsy was performed, the diagnosis of inspissated bile syndrome complicating hereditary spherocytosis would seem to be established beyond reasonable doubt, in view of the gradual disappearance of the obstructive jaundice and the eventual course of the anaemia. Yet the problems of diagnosis and management in the first 6 weeks of life were considerable. Faced with an ill, jaundiced and anaemic child, no haematological evidence of hereditary spherocytosis could be demonstrated. Haemolytic disease of the newborn due to rhesus or ABO incompatibility was ruled out, and atresia of the bile ducts could be excluded by the intermittent presence of urobilin in the urine and by the presence of bile-stained stools after intraduodenal instillation of magnesium sulphate. There was no evidence of septicaemia. The urine did not contain reducing substances; this excluded galactosaemia. Although congenital toxoplasmosis and cytomegalic inclusion disease could only be finally eliminated by liver biopsy, there was on the one hand no retinitis on fundoscopic examination and on the other no abnormal cells in the urine. There remained only neonatal hepatitis or haemolytic anaemia with superadded obstructive jaundice as likely alternatives.

Because of the reported beneficial effects of steroids in cases of 'idiopathic' inspissated bile syndrome,[3] a course of cortisone was given. Clinical improvement undoubtedly coincided with commencement of treatment but the significance of this is difficult to assess in view of the tendency to spontaneous improvement in this condition. Clinically and biochemically the liver appears to have suffered no lasting damage. Again, it is unfortunate that no liver biopsy was taken at the time of the splenectomy.

The more elaborate studies on red-cell fragility — incubated red-cell fragility, autohaemolysis and mechanical fragility[14]—were not performed in this critical early period. Although it has been held that the red-cell fragility may be normal at birth in cases of hereditary spherocytosis, the use of these recent techniques could prove this untrue. In 5 of 31 cases, however, the fragility using the standard method was normal when these cases were first examined and increased later.[8] In the present case the red-cell fragilities were repeated 1 year after splenectomy by Creed's method, using oxygenated heparinized blood, coupled with the thermal autohaemolysis test. It is of interest that no significant increase was detected compared with the control. Young *et al.*[14] found that in all of 11 patients with hereditary spherocytosis whom they investigated, the characteristic abnormalities of the erythrocyte persisted after splenectomy for the one or more years they were followed up.

The intraduodenal administration of magnesium sulphate as a cholagogue produced a definite response and was accompanied by no overt side-effects or diarrhoea. The use of parenteral bile acids would seem however to be safer and better.[15]

The importance of expectant treatment in this syndrome has been emphasized by Gellis *et al.*[3] who showed that mortality and morbidity were far commoner in cases explored surgically. The surgical management of neonatal obstructive jaundice has recently been reviewed by White.[16]

After subsidence of jaundice in the case here reported, the anaemia was severe enough to justify early splenectomy. This does not conform with the usual pattern of the disease in infancy.[9] Many infants have severe anaemia in the first few months of life, but, with transfusion therapy, the anaemia subsequently remains minimal. Splenectomy may therefore be deferred under a regime of careful observation. In view of the well-known danger of overwhelming infections in infants who have undergone splenectomy it is probably wise to delay the operation as long as possible.[8] This conclusion is however not supported by Laski and Macmillan[17] who claim to show that the incidence of serious infections in children after splenectomy is no higher than in a similar group after appendicectomy.

SUMMARY

Hereditary spherocytosis is a rare cause of jaundice in the neonatal period. A case complicated by the inspissated bile syndrome is described here. The obstruction cleared completely and splenectomy was later performed because of uncontrollable anaemia. The association of hereditary spherocytosis and the inspissated bile syndrome does not appear to have been previously reported.

I am indebted to Prof. S. S. Gellis of Boston, USA, for his advice, and to Dr. P. J. Barnard of Bulawayo, who kindly reviewed the draft of this article.

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THE PHYSICAL AND EMOTIONAL RESULTS OF HYSTERECTOMY

A REVIEW OF 162 CASES

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Our interest in the physical and emotional results of hysterectomy was stimulated by recent letters in the *British Medical Journal*[1,2] concerning the psychological preparation of patients for hysterectomy and especially by Dr. K. Dalton's dismal picture of the aftermath of the operation.[3]

In our practices we deal mainly with a rural community in the Eastern Cape consisting of wool, apple and citrus farmers and their families. This report is a follow-up of patients treated by hysterectomy during the last 5 years.

PROCEDURE

We explain to the patient and her husband pre-operatively, if possible, what the operation involves. Routine check-ups are made 6 weeks and 6 months after the operation, but for our present purpose we sent out simple questionnaires and asked for any further comments. The response was satisfactory and most patients appreciated the interest shown. A few, mostly those in the older age group, considered the questions about marital relations impertinent. One questionnaire was returned with the note: 'Your questionnaire is returned herewith. I regret my wife will not participate'. Many patients, however, wrote letters with their questionnaires, largely in the following vein: 'Geagte dokter, Ek is doodgelukkig, spekvet en so gesond soos 'n vis in die water'.

QUESTIONNAIRE

The questionnaire contained the following questions and the patients were asked to make a ring round the answer they considered correct in each instance:

1. How long was it before you were able to resume your normal duties?: *6 weeks, 3 months, 6 months.*
2. Is your general health: *better, same, worse?*
3. Are you satisfied with the results of your operation?: *yes, no.*
4. How do you feel about not having a flow of blood every month?: *pleased, unconcerned, displeased.*
5. How do you feel about not being able to fall pregnant? *pleased, unconcerned, displeased.*
6. Have you been told everything you want to know about the operation?: *yes, no.*
7. How much weight have you gained?: *under 10 lb., under 20 lb., under 30 lb.*
8. How soon did you resume marital relations?: *6 weeks, 3 months, 6 months.*
9. Has the operation made marital relations: *worse, better, same?*
10. Do you have any pain with intercourse?: *yes, no.*
11. Was intercourse satisfactory before operation?: *yes, no.*
12. What is your husband's attitude to your operation?: *pleased, unconcerned, displeased.*
13. What is the attitude of your friends to your operation?: *in favour, not in favour.*
14. Did you get hot flushes?: *yes, no.*
15. Did you benefit from treatment of the hot flushes?: *yes, no.*
16. Do you think headaches and depression worse than before the operation?: *yes, no.*

ANALYSIS OF REPLIES

We divided the patients into 3 groups: (1) those who had had total hysterectomy and bilateral salpingo-oophorectomy, (2) those who had had total hysterectomy but who remained with one or both ovaries, and (3) those who had had vaginal hysterectomy.

1. *Total Hysterectomy and Bilateral Salpingo-oophorectomy (108 Patients)*

Gynaecologists are still divided on the subject of removal of healthy ovaries in women after the age of 40 years. It has been our practice (influenced by opinion at the Chicago Lying-in Hospital, where one of us did a residency) to remove both ovaries after the age of 40. All these patients were in the over-40-years age group.

Question 1. Of the 108 patients in this group, 44 took up to 6 weeks, 60 up to 3 months and 4 up to 6 months to resume their normal duties. It seems that the average patient requires from 6 weeks to 3 months to resume normal duties.

Question 2. Two patients replied that their health was worse, and 106 that it was better. Therefore the operation, despite ablation of both ovaries, appears to be most successful in restoring a woman's health and even in improving it. We doubt whether the general health is adversely affected by castration, although this is contrary to the belief of many British gynaecologists.

Question 3. All the patients in this group were satisfied with the results of the operation, including those who maintained that their general health was worse. These replies are in direct contrast to those of Dr. Dalton who reported that, of her patients, 83% were satisfied less than a year after the operation and only 33% were satisfied after from 6 to 10 years. Our groups of patients had their operations between 6 months and 5 years before the questionnaire was sent out and we see no reason why this group of women, past the child-bearing age, need be expected to be unhappy about their operations.

Question 4 - 6. All these women were either pleased or unconcerned at being infertile and not menstruating. This was to be expected in this age group. Six patients replied that they had not been told all they wanted to know about the operation. Our records indicate that psychogenic factors played a large part even before the operation in these patients, but we feel that more should possibly have been done to explain the operation.

Question 7. Fifty-eight patients gained under 10 lb. in weight, 30 under 20 lb., and 20 under 30 lb. We feel that more stress should be laid on a normal diet in the post-operative months and the patients should be weighed more regularly.

Questions 8 - 11. Marital relations were of particular interest to us. Nearly 50% in this group replied that marital relations were not satisfactory before the operation. After the operation, 38% maintained that marital relations were improved, 47% that there was no change, and 15% said that they were worse. Of this 15%, half belonged to the group where relations were not satisfactory before the operation. We are of the opinion that the operation does little or nothing to mar the normal libido but we have been troubled by the complaint of lack of libido in general — a very common one. We are of the opinion that the lack of libido is so frequent that it must be considered a normal condition, and we believe this despite what most books on sex in the female have to say. We stress the need for coquetry and bluff as far as the husbands are concerned but have been disappointed with our results. Most women in this group resumed marital relations between 6 weeks and 3 months after operation and a common request at the 6 weeks' check-up is that we should say that intercourse should be postponed as long as possible.

Questions 12 and 13. Most husbands were pleased about the operation, some unconcerned and 6 displeased. This was rather surprising since we had anticipated that more husbands would be concerned. A few husbands asked whether their wives would be 'any good' after the operation. Except for a few who did not reply, all the patients stated that their friends were in favour of the operation. We have a strong impression that patients in this rural community are satisfied with the results.

Questions 14 and 15. Of these patients, 75% had hot flushes to some degree, and all but 12 benefited from treatment.

2. *Total Hysterectomy — One or Both Ovaries Remaining (36 Patients)*

Questions 1 - 5. This group resumed their normal activities from 6 weeks to 3 months after operation, and the general health of all except 1 was better — she replied 'the same'. All were satisfied with the results of the operation but 2 were displeased about the loss of their menstruation and child-bearing function. One unfortunate girl, a trained nurse, was only 21 years of age, and a hysterectomy had been advised by many gynaecologists to control her functional bleeding. The other was a divorced woman who had gross endometriosis and had already had 2 previous abdominal operations in which conservative surgery was done.

Questions 6 and 7. All replied that they had been told all they wanted to know about the operation. Most gained under 10 lb. in weight and 4 over 30 lb.

Questions 8 - 11. Marital relations were commenced in from 6 weeks to 3 months, but only 20 of this group commented on these questions. Six of these said relations were worse, 6 better and 8 the same. None of those who said they were worse complained of dyspareunia. Four stated that intercourse was satisfactory before the operation and 1 dissatisfied patient was having marital problems before the operation because of her husband's excessive drinking.

Questions 11 - 16. All the husbands were either pleased or unconcerned about the operation. The attitude of the patients' friends was largely in favour. Thirty patients had hot flushes, i.e. only 6 did not. Removal of the uterus no doubt interferes with the blood supply to the ovaries and is the most likely cause of the hot flushes. About a third of the patients thought headaches and depression were worse after the operation.

3. *Vaginal Hysterectomy and Anterior and Posterior Repair (18 Patients)*

The response to the questionnaire in this group was rather poor and only 18 out of 36 patients replied. They were largely over the age of 50 years and the questions caused a good deal of consternation to some patients, particularly those who were really old and widowed — and many were. The oldest was 90 years of age. The patients' own doctors kindly explained to them that only those questions which were applicable should be answered and that we were trying to ascertain the benefits of surgery.

Almost all had a marked degree of prolapse. The 18 who answered had returned to their usual activities in about 3 months. All said that their general health was better, and they were satisfied with the results of the operation. However, 1 patient, who takes care of her spastic adult son, said that she was well for 2½ years but that her prolapse had returned. None of them suffered from headaches. One patient presented with an enterocele 2 years after her vaginal hysterectomy. This was obviously missed at the first operation and has since been satisfactorily repaired.

CONCLUSIONS

We feel that, when a hysterectomy is performed for adequate indications and an effort is made to explain what the procedure involves, the results should be satisfactory. Pre-operative and postoperative advice and reassurance is most important and it is a wise move to warn the patient before operation about the probable onset of climacteric symptoms.

SUMMARY

Questionnaires were returned by 162 patients who had had an abdominal or vaginal hysterectomy. These questionnaires asked about their general health, their marital relations, and their own and their husbands' attitude to the operation. An overwhelming majority were completely satisfied with the results of the operation.

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43ste MEDIESE KONGRES (M.V.S.A.), KAAPSTAD, 24 - 30 SEPTEMBER 1961 : 43rd MEDICAL CONGRESS (M.A.S.A.), CAPE TOWN, 24 - 30 SEPTEMBER 1961

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6. *Early submission of manuscripts* will facilitate the arrangement of *combined sectional meetings* and the publication of the papers in roneoed book form, which will be made available at a nominal charge upon registration of delegates attending Congress.

7. *Papers of wide interest that may be used in combined sectional meetings are especially welcome.*

8. Papers accepted will be published in separate roneoed volumes devoted to the different sections. According to Congress rules the writer of any such paper shall not read his paper in full, but shall deal with special points in it by way of verbal communication lasting from 10 - 30 minutes depending upon the discretion of the Scientific Committee.

9. Papers submitted will be considered by the appropriate committee and a notification of acceptance will be sent to authors as early as possible. The Committee also has the right to suggest changes in the form and length of the papers.

10. *Copyright of all papers presented at Congress is vested in the 'South African Medical Journal'.* If an author desires his paper to be published elsewhere the permission of the Association's Editor must be obtained.

11. *All persons participating in the proceedings at Congress must be members of the Medical Association of South Africa. Overseas speakers and invited guests are eligible only if approved by the Scientific Committee.*

12. Papers sent by post should be *registered* to ensure safe arrival.

G. S. Muller Botha
Secretary, Scientific Committee

Congress Office
P.O. Box 643
Cape Town

INTERNATIONAL UNION AGAINST CANCER

SUB-COMMITTEE FOR AFRICA OF THE COMMITTEE ON GEOGRAPHICAL PATHOLOGY

The above Sub-Committee came into being in Johannesburg, South Africa, in 1957 under the Chairmanship of Prof. J. Gillman of the University of the Witwatersrand. It followed upon meetings at Kampala and Leopoldville in 1956 when the subject of demographic pathology in Africa with particular reference to liver disease was discussed by representatives from Europe, United States of America, and Africa.

The members of the Sub-Committee are interested in facilitating geographic pathology in general, but are particularly concerned with the demography of cancer in Africa.

Since its inception it has held one other meeting (at Leopoldville in 1959) and has concerned itself with:

1. The statistical aspects of cancer research in Africa.
2. The standardization of statistical methods for such research in underdeveloped communities.
3. The initiation and support of cancer surveys in different regions of Africa.
4. The relation of cancer in Africa to the biology of the African.
5. The investigation of nutrition, hepatic and endocrine function, soil analyses, socio-economic circumstances, and other factors in relation to cancer.
6. The preparation and publication of monographs on particular aspects of cancer in Africa.

In pursuance of its objects the Sub-Committee has sought to facilitate communication between research workers throughout Africa and, where possible, to assist them in their work. The Sub-Committee has no funds at its disposal for the support of research but it seeks, by making the proper contacts, to assist workers in obtaining support for their research activities. If cancer research workers in Africa feel that the Sub-Committee might usefully act as a type of clearing office, source of contacts, or advisory body, they are invited to communicate with the Secretary.

J. F. Murray
Hon. Secretary, Sub-Committee for Africa
U.I.C.C. (International Union against Cancer)
P.O. Box 1038
Johannesburg
4 January 1961

VRAE BEANTWOORD : QUESTIONS ANSWERED

ALLERGY TO GRISEOFULVIN

Q—*If, during the treatment of ringworm of the skin with one of the griseofulvin drugs, the patient becomes allergic to the drug, how should the scheme of treatment be changed?*

A—Although minor toxic or allergic side-effects occur quite frequently in the course of treatment with griseofulvin, no major or fatal cases of intolerance have yet been reported. Only rarely must treatment be abandoned because of intolerance to griseofulvin.

Headache, vertigo, gastro-intestinal upsets, arthralgia, periorbital oedema, urticaria, and erythematous and petechial rashes may be encountered. Depression of the leukocyte count is common, but agranulocytosis has not occurred. Inflammatory ringworm lesions, particularly vesiculo-pustular eruptions of the hands and feet, may worsen in a therapeutic reaction about the fifth day, but this is not an indication for suspension of treatment.

Headache occurs so often in the early days of treatment that patients should be warned of the possibility. Mild headache passes in a day or two and should be treated with aspirin. In the case of severe headache, treatment should be suspended until the headache has gone; then it can be recommenced with 1 tablet (250 mg.) the first day, 2 the second, 3 the third and finally back to the standard 4 tablets daily.

In the face of allergic reaction in a case where only griseofulvin is likely to be curative it would be reasonable, after the symptoms have subsided, to make an attempt at desensitization by giving first a very small dose and gradually increasing it if it is tolerated. If the allergic reaction recurs, griseofulvin will have to be abandoned and only topical fungicides used. It would naturally be pointless to try switching from one brand of griseofulvin to another because the active principle is the same in all.

Readers are invited to submit questions for this column to the Editor, *South African Medical Journal*, P.O. Box 643, Cape Town.

IN MEMORIAM

EMMANUEL JACOB SWIRSKY, L.R.C.P. & S. (EDIN.), L.R.F.P.S. (GLASG.)

Mr. J. E. Ellison, of Johannesburg, writes:

On 21 December 1960 Dr. E. J. Swirsky, a grand man of many parts; a doctor, humanitarian, and a gentleman, better known to a host of patients and colleagues as 'Mick', passed away suddenly in Tel Aviv, Israel. He was in his 64th year.

I write because he was a wonderful friend to me, in good times and in bad; a friend whom I venerated.

Dr. Swirsky qualified at Edinburgh University in 1921. While he was still studying he married Miss Sarah Silber and before he qualified his wife presented him with a son, who was later to follow in his father's foot-steps. On Dr. Swirsky's return to South Africa, in 1922, he settled in private practice for a few years in Morgenzon, Transvaal. He then moved to Johannesburg where he built up a large private practice with patients of every faith and many nationalities. He was also a railway medical officer and anaesthetist, a sphere in which he rendered invaluable services. His capacity for work was phenomenal and I think that what enabled him to do the work of more than two men so easily, was his remarkable clinical acumen and irrepressible sense of humour. Dr. Swirsky was beloved by his patients, whose welfare was paramount to him. To his colleagues he was a friend. In our long friendship I never heard him utter an unkind word. It was always a pleasure and an interesting experience to be associated with him in consultation for, not infrequently, with a wise remark and a twinkle in his eyes he would 'cut me down to size'. He was a good mixer, and a genial host.

Dr. Swirsky

Life did not always run smoothly for him. He had his good times, and set-backs, which he bore with a fortitude and courage rarely seen in any man. His son Sydney, who after qualification joined him in practice, was a great help to him, but he still worked like a Trojan. His daughter Faith was the apple of his eye.

On the reverse side, he lost his beloved wife about 6 years ago. He underwent 2 major operations, sustained 2 major fractures, at different times, with no complaint. He recovered from these, but became afflicted by an ailment which he knew would sooner or later encompass him. He did not allow this to get him down or wipe away his smile, but carried on. Not wishing to be an embarrassment or drain on his family, he decided about 4 years ago to emigrate to Israel and to devote himself entirely to anaesthetics. He eventually became the senior anaesthetist at the Assaf Harofe Hospital.

About a year ago he returned to spend a short holiday with his married daughter Faith, in Pretoria, with his son Sydney, and his brother and sisters, to whom we extend our deepest sympathy.

Eloquent testimony of the high esteem in which he was held in medical and lay circles, was borne out by the very large gathering and the glowing addresses which were delivered by leaders in various walks of life, at his graveside.

There are degrees of heroism and ranks of heroes. An attribute of perfect heroism is pure unselfishness, which is the most God-like quality in man. The more we live for others, the more in harmony are we with the divine power.

Good-bye Mick. Rest in peace.

IN DIE VERBYGAAN : PASSING EVENTS

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association will result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as those who change their addresses within the Union.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 30 January at 5.10 p.m. in the Institute Lecture Theatre. Dr. D. Ordman will speak on 'Weather, climate, life and Man'.

* * *

South African Society of Medical Women (M.A.S.A.), Cape Town Sub-Group. The Annual General Meeting of this Sub-Group will be held on Monday 30 January 1961 at 8.15 p.m. in the Gynaecology Lecture Theatre, Medical School, Observatory, Cape. During the evening Dr. M. B. Bennett will present a film dealing with the use of cytology in the diagnosis of gynaecological cancer. All women doctors are welcome. Refreshments will be served.

* * *

Dr. F. C. O. Allen, ortopediese chirurg, voorheen voltydse ortopediese chirurg aan die Algemene Hospitaal, Johannesburg, het op 1 Januarie 1961 in die praktyk getree saam met dr. G. G. Schepers te Oasim 201, Port Elizabeth. Telefoon 28281.

Mr. F. C. O. Allen, orthopaedic surgeon, formerly assistant orthopaedic surgeon at the Johannesburg General Hospital, joined Mr. G. G. Schepers in practice on 1 January 1961 at 201 Oasim, Port Elizabeth. Telephone 28281.

* * *

Dr. J. J. Frick, who has been doing a postgraduate course at the Louise Obici Hospital, in Virginia, USA, will return to South Africa at the end of February 1961. Until commencing practice, Dr. Frick may be contacted at Box 9, Malan Siding, District Wellington, Cape.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

GLUCOPHAGE

Westdene Products (Pty.) Ltd. announce the introduction of Glucophage, a new oral hypoglycaemic agent, and supply the following information:

Glucophage is N.N. dimethyldiguanide hydrochloride, which was discovered in the Research Department of Rona Laboratories Ltd. in France. Its mode of action is still incompletely understood, but it has been shown experimentally that it has a peripheral action, thus differing fundamentally from that of the sulphonylurea compounds. Toxic effects on blood, liver and kidneys do not occur and Glucophage is normally extremely well tolerated, although a few cases of digestive upsets, such as anorexia, nausea and diarrhoea in a mild form have been encountered. Reports indicate that side-effects are appreciably less than with phenethyldiguanide.

Glucophage has been used successfully in all forms of diabetes, even in severe cases with vascular degeneration. It is most effective in early and less acidotic diabetics and also in very obese patients. Long-standing or insulin-resistant diabetics, or cases where sulphonylureas have been ineffective, may respond to Glucophage. According to results in therapy with Glucophage in a random group of diabetics, success can be expected in 80 - 85% of patients.

Glucophage is slowly cumulative and the initial dose cannot therefore be used as a measure of susceptibility of the patient to therapy. Assessment should, in fact, be delayed until the 15th day. The dosage, which varies from ½ to 6 tablets per day, must be adjusted for each individual patient because individual sensitivity to this compound is variable.

Glucophage is now available in boxes of 30 or 500 tablets (500 mg.) packed in aluminium foil.

Further information may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

CORTIFLEXIOLE

Newport Trading Corporation (Pty.) Ltd. announce the introduction of Cortiflexiole, an oily suspension of hydrocortisone (0·4% free alcohol) and neomycin (1·15%), manufactured by Dr. Mann Laboratories, and supply the following information:

Cortiflexiole is a highly effective combination of the anti-phlogistic hormone, hydrocortisone, with the antibiotic neomycin for all topical use, except on mucosal membranes. It is superior in effect in all acutely inflamed and infected conditions of the skin and of the eye owing to a rapid and thorough absorption. Cortiflexiole also contains vitamin A to protect the epithelium.

The recommended administration is 2 - 3 applications per day.

Further information may be obtained from Newport Trading Corporation (Pty.) Ltd., P.O. Box 1871, Johannesburg.

BOEKBESPREKINGS : BOOK REVIEWS

STUDY OF BREAST-FED AND ARTIFICIALLY FED INFANTS

Weight Gains, Serum Protein Levels, and Health of Breast Fed and Artificially Fed Infants. A clinical and biochemical study based on 946 infants and children at the Mother's Hospital (Salvation Army) and the Queen Elizabeth Hospital for Children, London. Medical Research Council Special Report Series No. 296. By B. Levin, M.D., Ph.D.; H. M. M. Mackay, M.D., F.R.C.P.; C. A. Neill, M.D., M.R.C.P.; V. G. Oberholzer, B.Sc.; and T. P. Whitehead, F.R.I.C. Pp. x + 154, 69 figures. 16s. net. London: Her Majesty's Stationery Office. 1959.

This is the outcome of a longitudinal study which continued for 3½ years. Infants, full-time and premature, were under observation from birth and data were also obtained for older children. From this material a mass of information, written, tabulated and graphical, has been assembled.

Particular attention was given to the serum proteins, as a whole and as albumin and globulin. These are discussed in some detail and in relation to birth weight, age from conception, and actual age. The weight charts are similarly treated.

The association of weight gain and protein intake is demonstrated and the protein needs of a normal full-term infant are shown to be met by an intake of 0.85 g. per lb. per day, but prematures need more.

This is a worthy addition to the MRC Special Report Series. It is an essential addition to the reference library of every specialist paediatrician, biochemist, and medical school.

F.J.F.

DIURETIESE MIDDELS

The Physiological Basis of Diuretic Therapy. By R. F. Pitts, Ph.D., M.D. Pp. xiv + 332. 38 figures. 78s. Oxford: Blackwell Scientific Publications Ltd. Springfield, Ill.: Charles C. Thomas. 1959.

In die eerste deel van die boek word die volume, samestelling, en meganismes waarby homeostase van die liggaamsvloeistowwe teweeggebring word, sowel as die abnormaliteite wat in edeem aangetref word, behandel. 'n Uitstekende uitgebreide oorsig oor die fisiologie word deur die skrywer verstrek. Interessant en leersaam is die hoofstuk oor renale filtrasie, re-

absorpsie, en uitskeiding waar die fisiologiese meganisme in die lig van renale histologie (deur elektronmikroskopie verkry) gesien word.

'n Hoofstuk word afgestaan aan die regulering van volume en osmotiese konsentrasie, en die integrerende rol van die hormone wat verskillende sentra beheer, word uitgebeeld. Die eerste deel van die boek word afgesluit met 'n bespreking van die renale faktore wat verantwoordelik is vir die vorming van edeem. Die tweede deel gaan oor die meganisme en terapeutiese gebruik van diuretiese middels. 'n Uitgebreide uiteensetting van samestelling, werking, kontraindikasies, gevare, en gebruiksaanwysings van alle bekende diuretiese middels word uitgebeeld. Die voorafgaande fisiologiese uiteensetting vergemaklik die begrip oor werking van die diuretiese middels.

Die boek word aanbeveel vir alle nagraadse studente wat geïnteresseerd is in nierfisiologie en vir diegene wat 'n breër insig in diuretiese terapie wil bekom. C.L.W.

IMMUNIZATION IN CHILDHOOD

Proceedings of a Symposium on Immunization in Childhood. Pp. 139. 5 figures. 17s. 6d. net + 1s. 1d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1960.

This symposium was held in London in May 1959 in 5 sessions — risks of immunization, poliomyelitis and pertussis vaccination, combined prophylactics and other immunizations, programmes and recommendations. Each subject was introduced by a speaker of authority, and then discussed by the meeting. The whole makes most interesting reading, particularly those hazards of immunization, viz. provocation poliomyelitis, pertussis encephalopathy and convulsions, and faults in sterilization of instruments.

The importance of early inoculation against pertussis was stressed, some workers having started as early as the first week of life. This was done because the blood antibodies against pertussis are always low, and no doubt because it was felt that the infant could form antibodies at this early age, when the mortality is highest. On the other hand it was considered that poliomyelitis immunization was best delayed until 7 months of age to avoid interference by maternal antibodies.

The symposium agreed on two schedules for immunization, but the main preference appeared to be for Schedule B (set out below. Another schedule has been suggested in this *Journal*[1] (10 January 1959) and the whole matter still appears to be much of a matter of individual preference, and related to the epidemiological and economic features of different countries.

SCHEDULE B

Age	*Visit*	*Vaccine*	*Injection*	*Interval*
2-6 mths	1	Triple*	1	
	2	Triple*	2	4 wks or more
	3	Triple*	3	
7-10 mths	4	Poliomyelitis	4	
	5	Poliomyelitis	5	4 wks or more
15-18 mths	6	Triple*	6	4 wks or more
		Poliomyelitis†	7	
Smallpox some time during the first 5 years				
School entry	7	Diph. & tetanus	8	
8-9 yrs	8	Diph. & tetanus		
		Smallpox (re-vacc.)	9	
10-15 yrs	9	BCG	10	

* Diptheria, tetanus and pertussis.

† A 4th dose of poliomyelitis vaccine will be necessary, but its exact timing has not been decided.

1. Questions Answered (1959): S. Afr. Med. J., 33, 37. P.V.S.

MENTAL HEALTH OF STUDENTS

The Student and Mental Health. Edited by Daniel H. Funkenstein, M.D. Pp. xxv + 495. £1.15.0. net. London: H. K. Lewis & Co. Ltd. 1959.

This is a record of the First International Conference on Student Mental Health, which was held at Princeton in 1956 and was sponsored by the World Federation for Mental Health and the International Association of Universities. The main theme of the Conference, at which 10 countries were represented, was the promotion of mental health, in all its aspects, in colleges and universities.

As this book contains the opinions of a large number of authorities in this field, and as its scope is wide and its merits striking, it is impossible to mention in a review the individual sections in particular; each chapter is important and makes a significant contribution. At best one can mention a few points. The contributions include Eriksen's formulations on late adolescence; concepts of identity crises in psychosocial development; the conflicts and difficulties which affect the adolescent and which serve to make the period of study in a university less effective than it might be; and the function of the mental health worker in the university in the treatment of the disturbed student; as well as in bringing the principles of dynamic psychology and the other behavioural sciences to the awareness of staff and students.

The book serves to emphasize the realization that there can be few matters of greater importance for study and action than the problems of the large group of young men and women who go to colleges and universities to prepare themselves for their work in life; it also underlines how much needs to be done from the point of view of ensuring the greater stability and effectiveness of those who will often be leaders of the future.

Additional features for which the book deserves praise are an excellent summary, a series of recommendations, and an index.

This book is a valuable work and a credit to its editor. It is a *must* for all those who are concerned with the emotional components of late adolescence and education, and for those responsible for the administration of universities and colleges. H.M.

VOG IN DIE PARENTERALE HOLTES VAN DIE LIGGAAM

The Fluids of Parenteral Body Cavities. By Paul D. Hoeprich, M.D. and John R. Ward, M.D. Pp. iv + 98. Illustrations. $4·75. New York and London: Grune & Stratton, Inc. 1959.

Hoewel dié siektetoestande wat te doen het met vogophoping in die sereuse holtes van die liggaam, die sinoviale ruimtes, en die serebrospinale vogruimtes, klinies min te doen het met mekaar, word hulle tog in hierdie monografie saam bespreek vanweë hulle verband met vogversameling in die streke.

Die fisiese en biochemiese eienskappe van die voginhoud in hierdie verskillende ruimtes word in oënskou geneem. Daar is 'n uiteensetting van die normale anatomiese en fisiologiese aspekte van vogvorming in hierdie gebiede.

Daar volg ook 'n verklarende beskrywing van die kenmerke van vogophoping in hierdie holtes by verskillende siekteprosesse.

Die sereuse vog is naasteby 'n dialisaat van bloedplasma, terwyl dié in die gewrigsholtes 'n meer komplekse samestelling het met die byvoeging veral van musien. Die serebrospinale vog het 'n nog meer gekompliseerde ontstaan, want hier word die inhoud van verskillende stowwe in die vog verder bepaal deur die aktiewe sekresie deur die choroïdale pleksus en deur die meganisme van 'n bloed-serebrospinale vogversperring.

Hoewel dit vreemd voorkom om die uiteenlopende siektebeelde wat in verband staan met vogophoping in hierdie besondere kompartemente, saam te voeg in een oorsig, vind 'n mens dit nogtans 'n baie praktiese en waardevolle samevatting.

Daar word ook praktiese wenke aangebied en goeie aanduidings gegee hoe om die vog te verkry, te ondersoek, en die bevindings te interpreteer. A.J.B.

PYELONEPHRITIS

Pyelonephritis. By Fletcher H. Colby, M.D. Pp. vii + 232. 95 illustrations. 60s. + 2s. 9d. postage. Baltimore: The Williams and Wilkins Company. London: Ballière, Tindall & Cox Ltd. 1959.

The frequency and the gravity of pyelonephritis is attested by the numerous reports that have appeared in recent years.

This is a lavish little monograph written by a urologist

who states that the book should be useful for students and general practitioners, but it is doubtful whether many will be prepared to pay 62s. 9d. — the price of this work.

This book represents a conventional but by no means comprehensive account of the aetiology and clinical features and treatment of the condition. It does, however, include a section on pyelonephritis in infancy and childhood and the relationship of diabetes mellitus, pregnancy, and hypertension to the disorder are each considered separately. It has the merit of reflecting the author's personal experience and contains valuable information from the records of the Massachusetts General Hospital. There are defects such as the lack of critical appraisal of reports of the reputed high incidence of the condition — all old scarring need not necessarily indicate chronic pyelonephritis. The sections on anatomy are not really necessary. The discussion on renal function is elementary, while studies of the functional aberrations of pyelonephritis receive scant attention. There are many very good illustrations of pyelograms, but they are unnecessarily numerous, particularly in the section on chronic pyelonephritis.

The advice on limitation of instrumentation to those patients in which it is clearly indicated, is to be endorsed. The dangers of catheterization are considered but, while advocating clean-voided urine specimens for the male, in speaking of voiding specimens in the female he states 'all seem complicated and their technique undignified'. Indignity is a poor reason for abandoning this procedure. L.E.

BIBLIOGRAPHY OF CHEMICAL CARCINOGENESIS

Bibliography of Cancer Produced by Pure Chemical Compounds. A survey of the literature up to and including 1947. By Otto Neubauer, M.D. Pp. xxvii + 604. London: Oxford University Press. English price 42s. 1959.

This bibliography contains the papers published on carcinogenesis by pure chemical substances up to and including the year 1947. There is a subject index, with references to 4,960 papers, subdivided into 32 sections dealing with various aspects of carcinogens, such as their chemical and physical properties, administration, effects, and intrinsic and extrinsic influences. The sections are subdivided according to the chemical nature of the carcinogen. The papers are arranged chronologically. There is also an author index, with many details, each entry being followed by a number or series of numbers, which indicate the locations in the subject index. There is also a list of tumour sites and special kinds of tumour.

Cancer research, which calls in the aid of many sciences, has provided an enormous literature, which requires to be catalogued, classified, and indexed. This bibliography records work done in a field not covered by other compilations of this nature. It will be of great help to those engaged in cancer research, and also to chemists, industrial scientists, and experimental pathologists.

RÖNTGENDIAGNOSE VAN DIE SPYSVERTERINGS-KANAAL

Röntgendiagnostik des Magen-Darmkanals. Von Prof. Dr. R. Prévôt und Priv.-Doz. Dr. M. A. Lassrich. xii + 346 Seiten. 544 Abbildungen. Ganzleinen DM 119.00. Stuttgart: Georg Thieme Verlag. 1959.

Die vraag kan miskien gestel word of daar op die huidige stadium plek is vir nog 'n handboek oor die röntgendiagnose van die spysverteringskanaal. As so 'n boek die nuutste werk insluit, of nuwe lig werp op vorige opvattings, dan is die antwoord ja. Gemeet aan die eerste vereiste, is daar sekerlik plek vir hierdie werk.

Daar is byvoorbeeld 'n verwysing na Roviralta se opvatting oor hiatusbreuk by suigelinge, wat iets van die jongste tyd is. Oor prolaps van maagslymvlies in die duodenum is daar 'n interessante bespreking, hoewel die skrywers nog nie die graad van prolaps koördineer met die mate van saamtrekking van die canalis egestorius nie, soos in baie gevalle gesien word. Die neonatale gasinhoud van die dermkanaal is 'n onderwerp wat bespreek word, en so meer.

Een van die belangrikste hoofstukke gaan oor die verhouding tussen die sg. gastritisbeeld en karsinoom. Aan die hand van talryke gevalle word bewys hoe 'n sg. gastritis na 5 - 15 jaar in 'n karsinoom kan ontwikkel. In ons ondervinding is dit die eerste handboek wat hierdie punt so pertinent stel, en dit sal röntgenoloë sekerlik beïnvloed om baie meer klem te lê op opvolgingsondersoeke waar 'n pasiënt met vae bobuikklagte 'n sg. gastritisbeeld wys. Slegs oor hierdie hoofstuk verdien die boek sy bestaansreg.

Die hoofstuk oor die post-operatiewe maag is besonder geslaag. Dit sluit skematiese voorstellings in van die verskillende operasies, wat seker 'n aanwins is. In 'n oogopslag kan nou gesien word hoe Mikulicz-Krönlein die Billroth II gedemonstreer het.

Die illustrasies het deurgaans 'n puik kontras en definisie. Hier het die skrywers sekerlik hulle eie stelling gevolg, nl. dat in geen afdeling van die röntgenologie die diagnose soveel afhang van die kwaliteit van die ondersoek as in die spysverteringskanaal nie.

Aan die debietkant kan genoem word dat Torgersen se fundamentele navorsing oor die spierstruktuur van die canalis egestorius weer eens oor die hoof gesien is. 'n Mens kan nie die prepiloriese saamtrekkings op 'n intelligente manier verklaar sonder verwysing hierna nie. 'n Jaar of twee gelede nog het McNaught, van Edinburg, Torgersen se bevindings deur sy eie disseksies bevestig.

Vir naslaandoeleindes sou dit beter gewees het as die literatuurlys elke hoofstuk gevolg het, in plaas van om dit in 20 bladsye aan die end van die boek op te hoop. 'n Duidelike literatuuroorsig, lyk dit, is iets wat nou eenmaal te veel is vir kontinentale skrywers.

'n Boek wat Forsell getrou volg en beweer dat morfologiese veranderinge van die slymvlies met die helderheid van 'n anatomiese preparaat weergegee kan word, nie deur willekeurige opnames nie, maar deur 'n goed ontwikkelde, moeitevolle deurligtingstegniek, is sekerlik sy sout werd.

A.D.K.

RADIOLOGY OF THE SMALL INTESTINE

Radiologic Examination of the Small Intestine. 2nd edition. By R. Golden, M.D. Pp. xxv + 560. 176 figures. £11 8s. Oxford: Blackwell Scientific Publications Ltd. Springfield, Ill.: Charles C. Thomas. 1959.

The first edition of this book rapidly became a standard reference. The second will do the same. It has more than twice as many illustrations, much enhanced by the use of an art paper, and it is justifiably twice as long.

The absence of illustrated definitions of many of the terms, for example flocculation and segmentation, is a major defect that is confusing. A minor defect is the imbalance of the section on intussusception. Here the emphasis is on functional intussusception, and the commoner types are not fully dealt with. The inexperienced reader may be left with the dangerous impression that a barium meal is a usual method of examining for intussusception.

The very useful chapter on post-operative states omits reference to the dumping and the afferent loop syndromes.

These minor deficiencies are more than counterbalanced by the full and detailed chapters on the other aspects of radiology of the small bowel. This volume can be recommended to radiologists, student radiologists, and physicians. H.J.

DERMATOLOGY

Dermatologie und Venerologie. Einschliesslich Berufskrankheiten, dermatologischer Kosmetik und Andrologie. In 5 Bänden. (Einzelne Bände erscheinen in zwei Teilen.) Herausgegeben von Prof. Dr. Dr. h.c. H. A. Gottron und Prof. Dr. Dr. h.c. W. Schönfeld. 20% ermässigter Subskriptionspreis für das Gesamtwerk. Der Subskriptionspreis gilt bis zum Erscheinen des letzten Bandes. Jeder Band ist einzeln zum Ladenpreis käuflich. *Band III, Teil 2. Krankheiten der Hautanhangsgebilde einschliesslich Akne und Rosacea — Hautkrankheiten bei inneren Störungen* (Mit Sach- und Namenregister für Band III, Teil 1 und 2). Pp. xiv + 706. 317 zum Teil farbige Abbildungen. Ganzleinen DM178.-. Subskriptionspreis DM142.40. (Der Bezug von Teil 1 — erscheinen im Juni 1959 — verpflichtete zur Abnahme von Teil 2). Band III (in zwei Teilen) Ganzleinen DM343.-. Subskriptionspreis DM274.40. Stuttgart: Georg Thieme Verlag. 1959.

Part 2 of volume 3 of Gottron and Schönfeld's authoritative

work is concerned largely with skin changes associated with general bodily disturbances such as liver, pancreas and intestinal disease, as well as the dermatological manifestations of avitaminosis and deficiencies of various metals and trace elements. Professors Tappeiner and Wodmansky, of Vienna, contribute a comprehensive description of those metabolic diseases, the lipoidoses, the xanthematoses, amyloidosis, hyalinosis, mucinosis, etc., the clue to which at times is revealed by characteristic skin signs. This section commands especial attention by its very completeness of textual description and the informative illustration of actual cases. It is, of course, up to date and well documented. Even the rare condition ochronosis, with its distinctive triad or symptoms — black urine, yellowish pigmented cartilage and osteo-arthritis — receives detailed attention.

Diseases of the hair and nails are fully discussed in separate chapters. That on the hair comprises 100 pages with several pages of references, and is itself a monograph on the subject. The illustrations, including an excellent one of trichoptilosis — axial splitting of the hair — are of a very high standard, conveying an accurate distinctive picture. The classification, too, is sensible and helpful, enabling one to forecast a prognosis based on pathology. The therapeutical recommendations are restrained and entirely unsensational.

Functional disturbances and diseases of the sweat glands and sebaceous glands each receive attention in separate chapters and by different contributors. Acne vulgaris comes under discussion here and rosacea (without the qualifying word acne!) but, regrettably, no new light is shed either on the aetiology or successful treatment except that favourable mention is made of superficial X-ray therapy. Hormone preparations are not wholeheartedly recommended.

Pruritus is a symptom for which patients are frequently referred to the dermatologist. More than 20 pages of text are devoted to a close examination of various pathological conditions, internal and external influences, dysfunction of internal organs, and the manifold other factors which bring about a generalized itch. The number of references to the literature runs into several hundred.

Nothing but praise can be given to the physical appearance, printing and binding. The royal-blue covers with gold lettering, preserved under an attractive dust jacket, make a fitting setting for this praiseworthy addition to a work that has already received universal acclaim. C.K. O'M.

SURGICAL NURSING

Surgical Nursing and After-treatment. 11th edition. By T. Edward Wilson, M.D., M.S., M.Sc.(Melb.), M.R.A.C.P., F.R.C.S. (Eng.), F.R.C.S. (Edin.), F.A.C.S., F.R.A.C.S. Pp. x + 618. 361 illustrations. English price 30s. net. London: J. & A. Churchill Ltd. 1960.

This is the 11th edition of the late Rutherford Darling's well-known text-book for nurses; but on this occasion his former cooperator, Mr. T. Edward Wilson, has undertaken the revision necessary since the last edition, which was published about 9 years ago.

New sections have been devoted to the nursing treatment arising from new techniques in the surgery of the heart, lungs, oesophagus and adrenal glands, and new chapters have been added on the nursing aspects of anaesthesia and the care of the unconscious patient, as well as the care of patients before and after radiotherapy.

A number of other important revisions have been made which have improved an already good book, and the whole achieves its original purpose of seeking to make the nurse an essential and competent member of the surgical team. A.H.T.

SEWAGE POLLUTION OF BATHING BEACHES

Sewage Contamination of Bathing Beaches in England and Wales. Medical Research Council Memorandum No. 37. Pp. iv + 23. 2s. 6d. net. London: Her Majesty's Stationery Office. 1959.

This small treatise of 23 pages, published by the Medical Research Council of Great Britain, is primarily devised for the non-medical reader. The technical details always inseperable from a publication of this type are, however, available for medical and bacteriological specialists in the *Journal of Hygiene* of December 1959.

The historical background and all available information on the alleged health risks of bathing in sewage-polluted coastal waters have been most satisfactorily and exhaustively sifted. Only one outbreak of typhoid fever — which occurred as far back as 1908 in the Royal Marine Depot in Walmer, Kent — has, on epidemiological grounds, been ascribed to bathing on a beach which was being polluted by a sewage outfall.

Innumerable bacteriological examinations of sea-water, collected at different phases of tide and wind, have been carried out in various parts of the country in an effort to correlate these findings with the incidence of diseases such as poliomyelitis and the gastro-intestinal infections.

It is suggested that comminutors could with advantage be installed at sewage outfalls with a view to pulverizing the sewage and so exposing it to the bacteriostatic effect of the sea-water.

All in all, the Commission finally comes to the conclusion that, apart from the aesthetically revolting sewage-polluted beaches, which definitely should not be used, there is, on the available evidence, little or no risk to health from bathing in sewage-contaminated sea-water.

There is a growing apprehension on the part of the public regarding the danger of the spread of poliomyelitis and other intestinal infections as a possible result of bathing in sea-water into which a sewage outfall discharges; and it is therefore most desirable that this publication should be closely studied by all persons who are responsible for the public health or interested in the subject. E.D.C.

THE BRITISH FORMULARY

British National Formulary 1960. Standard edition. Published jointly by the British Medical Association and the Pharmaceutical Society of Great Britain. Pp. 272. English price 7s. 6d., interleaved 11s. 6d. Obtainable from A.P.S. Journal (Pty.) Ltd., P.O. Box 6290, Johannesburg. London: The Pharmaceutical Press. 1960.

This handy pocket volume, with more pages than the previous edition, has not undergone any major reconstruction. The same policy has been maintained to keep this prescribers' formulary convenient in size and comprehensive. Formulae are still included if they are widely used, but some have been deleted. There is a section providing the formulae of preparations for infants and children. Many pages have been devoted to a useful comprehensive glossary of proprietary or trade names and the equivalent non-proprietary preparations with analogous therapeutic effects. In this, the standard edition, there are introductory sections with brief description of various groups of drugs — analgesics, antacids, anthelmintics, antibiotics, and so forth. There are notes on other topics that are useful to prescribers. The volume will be found to be of great assistance in practice and in hospital. Distribution to clinical students would also assist in the encouragement of rational prescribing, with corresponding decrease in hospital and national drug expenditure. N.S.

DERMATOLOGY IN PAEDIATRICS

Pediatric Dermatology. By Henry H. Perlman, M.D., Phar. D. Pp. xii + 477. 203 figures. $18.00. Chicago: Year Book Publishers, Inc. 1960.

Perlman has written a book on dermatology which will make its greatest appeal to the practising paediatrician in search of a single self-contained review of the subject. It is doubtful whether the dermatologist will find enough unfamiliar material within its covers to justify the outlay, or the general practitioner either, who will probably be better off with a work on skin disease covering all age-groups. The reviewer finds the work imprecise, and it seems that its writer is acting mainly as a transmitter of the opinions expressed by other Americans, little altered by his own clinical experience or discrimination. All adverse criticism of the book which one may wish to make can be reduced to these causes. Otherwise it is an attractively produced work in the best tradition of American scientific texts. G.H.F.